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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 20 JULY 1934 NUMBER 1

MÉNIÈRE'S DISEASE

SYMPTOMS, OBJECTIVE FINDINGS AND TREATMENT IN FORTY-TWO CASES

WALTER E. DANDY, M.D.

BALTIMORE

In earlier publications [1] I have shown that Ménière's disease may be permanently cured, with scarcely any risk to life or function. In the entire series which I studied there was no death, and, except in two early cases in which the facial nerve was injured, there was no complication. With the more recent hemisection of the auditory nerve, instead of total section, even the loss of residual hearing—the only function affected by total section and usually of little practical value—may be avoided. In no case has there been, so far as I know, a single attack of dizziness subsequent to the operation.

With the advent of partial section of the auditory nerve (i. e., total section of the vestibular branch and preservation of the cochlear branch), one is naturally led to wonder whether, if this form of treatment were applied in the early stages of the disease, it might not be possible to cure the dizzy attacks and at the same time make possible the retention of normal or nearly normal hearing. Since the underlying etiology and pathology of Ménière's disease are not known, it is impossible to predict that the elimination of the dizzy spells will prevent the progressive loss of hearing that occurs in the natural progress of the disease. The answer must await the verdict of time. At least there is nothing to lose and everything to gain by early treatment. Although spontaneous cures do occur in Ménière's disease, they are probably rare. The occasional long intervals between attacks often mislead the victims into a feeling of false security and into the hope or assumption of cure. The detailed analysis of the symptoms of Ménière's disease was undertaken largely to determine (1) whether early diagnosis is possible before

1. Dandy, W. E.: (*a*) Ménière's Disease: Its Diagnosis and a Method of Treatment, Arch. Surg. **16**:1127 (June) 1928; (*b*) Ménière's Disease (Aural Vertigo), in Lewis, Dean: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1933, vol. 12, chap. 1, p. 207. (*c*) The Diagnosis and Treatment of Ménière's Disease, Tr. Am. Therap. Soc., 1932; (*d*) Ménière's Disease: Diagnosis and Treatment; Report of 30 Cases, Am. J. Surg. **20**:693, 1933; (*e*) Treatment of Ménière's Disease by Section of Only the Vestibular Portion of the Acoustic Nerve, Bull. Johns Hopkins Hosp. **53**:52, 1933; (*f*) The Effect of Hemisection of the Cochlear Branch of the Human Auditory Nerve, Bull. Johns Hopkins Hosp. **54**:208, 1934.

appreciable or marked loss of hearing results and (2) whether the similar attacks of pseudo-Ménière's disease, differing from Ménière's disease only in the absence of unilateral defective hearing and tinnitus, may not be an early stage of true Ménière's disease. However, I must not leave the impression that partial section of the auditory nerve is an absolute cure for Ménière's disease. Symptoms following this procedure have been absent for only thirteen, seven and four months, respectively, in the three cases in which this operation was performed—obviously too short a period for positive proof—but it is difficult to see how attacks of dizziness can recur when the vestibular branch of the nerve is totally severed (the cochlear branch only remaining).

Little need be said concerning the diagnosis of Ménière's disease in its characteristic form; there are surprisingly few departures from the syndrome of sudden terrific dizzy attacks, accompanied by nausea and vomiting, plus deafness and tinnitus in the affected ear. Numerous other symptoms may be added, but the basic syndrome is almost invariable.

So devastating are the attacks that patients live in constant horror of their recurrence. They are, of course, prevented from working where the sudden fall might cause disaster; many of the pleasures of life are denied them, and all too frequently they shun society, remaining at home where they may be, in a degree at least, better able to withstand the attacks, which demand immediate attention. Jonathan (Dean) Swift, the British satirist of the eighteenth century, and England's great neurologist, Hughlings Jackson, were victims of Ménière's disease during much of their lives. A distinguished American man of letters, Gamaliel Bradford, was also a victim. Throughout the last twenty-five years of his life there was little relief from the recurring attacks, which he described in his "Journal" [2] and so aptly called "the swoop of the hawks."

FREQUENCY

The relative frequency of Ménière's disease will doubtless be determined with greater accuracy two or three decades hence. At present the diagnosis is all too frequently missed, and in addition lack of knowledge concerning the value of operative treatment has prevented the same concentration of cases that obtains in other well recognized conditions with a definite form of treatment. One can say, at least, that Ménière's disease is common—perhaps one-fourth or one-fifth as frequent as trigeminal neuralgia—though it is not improbable that in time these figures may be halved.

ANALYSIS OF DATA

Age.—Of the series of forty-two patients studied only one came for treatment before the thirtieth year. In two thirds of the cases the

2. Published by Houghton Mifflin Company, Boston, 1933.

disease appeared in the fifth and sixth decade. However, when one inquires into the time of onset, it is found that in only one case did the attacks begin before the twentieth, and in only one after the sixtieth, year. Although the disease begins somewhat less frequently in the third decade, the time of onset is spread evenly over the succeeding three periods. Ménière's disease rarely begins in youth or in extreme old age. Usually it occurs in healthy persons and without any apparent underlying cause. The attacks, although of great severity, do not cause death or shorten life. The time of onset closely resembles that of trigeminal tic douloureux.

Sex.—Twenty-six cases (62 per cent) occurred in males and sixteen (38 per cent) in females. These figures would appear to indicate a

TABLE 1.—*Age of Patients at Beginning of Treatment and at Onset*

	10-20 Years	20-30 Years	30-40 Years	40-50 Years	50-60 Years	60+ Years
Age when patients came for treatment	..	1	7	15	13	6
Age at onset of Ménière's disease	1 (12 years)	6	11	12	11	1

TABLE 2.—*Symptoms at Onset*

	Dizziness	Deafness	Tinnitus	Dizziness and Deafness	Dizziness and Tinnitus	Tinnitus and Deafness	Dizziness, Deafness and Tinnitus
Number of cases	8	8	10	3	1	8	4

predilection for the male sex. However, I have no explanation to offer in the support of such a possible predilection.

Side of the Lesion.—There is a curious disproportion in the incidence of Ménière's disease on the two sides; the disorder was on the left, in twenty-nine (70 per cent) of the patients studied, and on the right in only thirteen (30 per cent). If this difference is significant, no explanation is as yet apparent.

In table 2 it may be seen that the three conspicuous symptoms—dizziness, deafness and tinnitus—occurred at the onset in about equal degree. In three cases dizziness and deafness, in eight cases tinnitus and deafness, in one case dizziness and tinnitus, and in four cases all three disturbances, occurred synchronously as the initial symptoms of this disease.

Nausea and vomiting were so clearly related to the dizziness that their time of onset practically coincided with that of the dizziness.

Deafness.—In every case of Ménière's disease, as otolaryngologists now understand it, there is loss of hearing on the side of the lesion;

this is the outstanding objective sign. The deafness is usually, but not always, gradually progressive, and at the time when the patient appears for operation the hearing is rarely of much use. In one instance—case 23—the patient insisted that the loss of hearing developed suddenly during a period of twenty-four hours.

The relationship in regard to time between the onset of deafness and that of the two other major symptoms, namely, tinnitus and dizziness, is variable; for example, in eleven cases deafness occurred before tinnitus; in thirteen cases it occurred after tinnitus, and in fourteen cases these symptoms occurred synchronously. Deafness may antedate tinnitus by periods varying from one month (case 27) to twenty years or more (case 22). In three cases it antedated tinnitus by a few months, and in four, by more than twenty years. On the other hand, tinnitus may antedate deafness by from a short interval (case 1) to sixteen years (case 7). The relationship of deafness to the dizzy attacks is much the same in regard to time of onset. In nineteen cases deafness preceded the dizzy attacks by from one month (case 27 and 30) to thirty-eight years (case 18). In twelve cases dizziness antedated deafness from six months (case 5) to twenty years (case 20). In eleven cases deafness and dizziness occurred together in the beginning attacks.

Tinnitus.—Tinnitus is typically confined to the involved side, but at times it may be bilateral, as in cases 20, 21, 24, 31, 32 and 40. Tinnitus is variously described as a roaring, buzzing, whistling, hissing noise, a ringing sound like escaping steam or a noise made by machinery. Usually it is constant, but this is not always true; there may be free intervals of varying time. In case 3 the tinnitus was only occasional. In case 14—the only one of the series to show this feature—there was none. In case 7, although there was a return of dizzy spells after a free interval of seventeen years, the tinnitus persisted in the interim. In case 40 the tinnitus was constant for seventeen years, then disappeared for a time. In case 23 tinnitus was constant for several years and then became intermittent.

It is interesting that in eighteen cases tinnitus appeared before dizziness (from a few weeks to many years). In cases 23, 21, 38, 6 and 18 tinnitus preceded dizziness by four and one-half, twelve, thirteen, twenty-three and thirty-eight years, respectively. In eight cases dizziness antedated tinnitus by equally variable periods of time: six months (case 10), one year (cases 9 and 16), five years (case 29), eight years (case 41), ten years (case 19), eighteen years (case 39) and twenty years (case 20).

The change in the character of the tinnitus during the attacks is of interest. It may be increased before (one case), during (ten cases) or after (two cases) the attacks; there was no change in seven cases. Usually the tinnitus is intensified, or the pitch may be changed. In case

Case	Na Pa Ye O	
1	M. 19	
2	J. 19	
3	R. 19	
4	L. 19	
5	F. 19	
6	H. 19	
7	A. 19	
8	M. 19	
9	A. 19	
10	W. 19	
11	A. 19	
12	W 19	
13	M. 19	
14	H. 19	
15	J. 19	

TABLE 3.—*Synopsis of Forty-Two Cases*

	Name of Patient and Year of Operation	Age (Yrs.) and Sex	Side	First Symptom	Duration of: Attacks	Dizziness	Deafness	Tinnitus	Nausea	Vomiting	Frequency of Attacks	Duration of Each Attack	Longest Free Interval	Is Tinnitus Influenced by Attacks?	Character of Dizziness	Loss of Hearing with Audiometer Test
	M. C. F. 1927	55 F	Right	Tinnitus	18 months	18 months	18 months	Longer than attacks	18 months	18 months	Almost constant for last month; average, 1 per week	Several hours	6 weeks	.....	Objects rotate; no constant direction	Marked
	J. H. 1930	46 F	Left	Tinnitus	6 months	6 months	6 months	Several years	6 months	6 months	2 per week	From 1 minute to 1 hour	1 month	Cannot recall	.....	Gradually progressive; subtotal
	R. K. 1931	48 M	Right	Tinnitus	2 years	2 years	2 years	10 years; occasional	2 years	2 years	1 or 2 per week	From 3 to 4 hours	6 months	No	.....	Slight
	L. H. 1928	47 F	Left	Deafness	5 years	5 years	5 years	5 years	5 years; none for the past 3 years	5 years; none for the past 3 years	1 or 2 per month	From 3 to 5 hours	2 years	Increased during attacks; less after	Objects turn	Subtotal on left
	F. B. G. 1929	37 M	Left	Tinnitus	9 months	9 months	3 months	18 months; persistent	In first attack only	In first attack only	1 or 2 per week	From 1 to 3 hours	1 month	No	Objects turn	Subtotal on left
	H. B. 1931	55 M	Left	Deafness and tinnitus	10 months	10 months	24 years (?)	24 years	Occasionally	Occasionally	2 or 4 per week	From ½ hour to 2 hours	.....	Increased before	Objects turn always same way	Almost total
	A. E. 1930	47 F	Left	Tinnitus	18 years; 1 year	18 years; 1 year	2 years; rapidly progressive	18 years; constant	18 years; 1 year	18 years; 1 year severe	2 or 3 per week; nearly every day shortly before operation	From ½ hour to 48 hours	16 years	No	Objects turn and are blurred	Subtotal
	M. E. R. 1932	56 F	Left	Deafness	3 weeks	3 weeks	2 years	3 weeks	Only once	None	1 per week	From ½ hour to 2 hours	1 week	Increased before	Objects turn like a top	Subtotal
	A. U. 1927	32 F	Left	Dizziness	2½ years	2½ years	20 months	18 months	2½ years	2½ years	3 per week; increased in frequency	From ½ hour to 3 hours	2 months	Increased during attack	Objects moving	65% loss (18% loss in good ear)
	W. D. P. 1927	47 M	Left	Dizziness	1 year	1 year	6 months	6 months	None	1 year	1 per month	Up to 3 weeks	4 months	Increased during attack	Objects rotate	Almost total
	A. H. 1927	34 F	Left	Dizziness and tinnitus	15 months	15 months	15 months	15 months	15 months	15 months	1 or 2 per month	From 1 to 2 hours	2 months	Increased during attack	Objects turn to right	Subtotal
	W. E. S. 1931	61 M	Left	Deafness	5 years	5 years	7 years	5 years	5+ years	5 years	3 attacks within 10 days; respite of 5 years	A few hours	5 years	.....	Objects rotate	Slight 5 years before operation; moderate at time of operation; but marked decrease
	M. T. 1927	54 F	Left	Dizziness	6 years	6 years	.....	.....	6 years	6 years	2 or 3 per month; several a day at time of operation	From ½ hour to 8 hours	1 year	Increased during attack	Objects turn to left	75% loss; also 30% loss in other ear
	H. N. 1928	35 M	Right	Dizziness and deafness	3 years	3 years	3 years	None	3 years; none in attacks shortly before operation	3 years; none in attacks shortly before operation	About 1 per month	Up to 3 days	2 months	Increased before and during attack; less after	Objects turn	Only slight hearing remains
	J. R. H. 1929	43 M	Left	Deafness	2 years	2 years	10 years	Buzzing [illegible] 2 years	2 years	2 years; absent shortly before operation	Several every week; increased	Up to several hours; average, 1 hour	3 months	.....	Objects turn to right	Subtotal
16	E. R. 1929	41 M	Left	Dizziness and deafness	1½ years	1½ years	1½ years	6 months	None	In one attack only	1 per week	Up to 2 hours	9 months	Increased during attack	Objects turn clockwise	Subtotal
17	C. H. 1930	57 M	Left	Deafness	3 years	3 years	3 years	Constant for 3 years	3 years	3 years	1 per month	From 3 to 6 hours	6 months	Increased during attack	Objects rotate	Moderate; also some loss in other ear
18	L. F. B. 1930	69 M	Right	Tinnitus	2 years	2 years	40 years	40 years	Only in most severe attacks	Only in most severe attacks	1 per week; maximum, 3 per day	3 hours	.....	Not at all	Objects rise up in front of patient	Few tones remain
19	M. T. 1930	38 F	Right	Dizziness	20 years	20 years	10 years	10 years	20 years	20 years	1 or 2 per week; formerly 1 per year	From 15 minutes to ½ day; average 2 hours	1 year	Increased during attack	Worse when lying, especially on back	Marked; also loss for high tones in other ear
20	A. L. 1930	45 [illegible]	Right	Dizziness	30 years	30 years	10 years; [illegible]	10 years; [illegible]	30 years	30 years	2 or 3 per [illegible]	From 1 to [illegible]	12 years	Increased [illegible]	Objects ro- [illegible]	Marked; slight [illegible]

Vestibular Function	Other Symptoms	Previous Operative Procedures Based on Wrong Diagnosis	Anesthetic	Postoperative Dizziness on Turning Head: Immediate	Postoperative Dizziness on Turning Head: Late	Does Tinnitus Persist?	Is There Any Possible Cause of Attacks?	Comment
Normal	Some ringing in other ear shortly before operation; bradycardia (60) in attacks	Gallbladder and appendix removed	Local	No	No	No	None	
Not tested	Tingling sensation around left ear and neck preceded attacks; cancer of breast removed 3 years before operation	Gallbladder removed	Avertin	6 months	No	Yes	Measles and aural infection	
Total loss on right	Warm, flushed feeling in affected ear; slight lateral nystagmus; vomiting caused improvement in attacks; vomiting only at times; patient felt better lying on side than on back during attacks; slightly dizzy 12 to 24 hours after attack	.....	Local	10 days	No	No	.....	Internal auditory artery bent and dislocated facial nerve; auditory nerve probably not affected
Total loss on left	Corneal reflex absent on affected side; positive Romberg sign on left; patient weak and miserable for several days after attacks; variation in severity of attacks; "falling spells" preceded dizziness; patient lay flat on back to improve dizziness	Tonsillectomy	Ether	No	No	Less	.....	
Response to caloric test less acute	Sight nystamus to either side; patient unable to walk for 3 days after one attack; transient feeling of dizziness on turning head	.....	Rectal, ether	6 months	No	Never changed	.....	
Normal	Attacks benefited by lying on left side; marked loss of hearing in opposite ear	.....	Local	No	No	Never changed	Patient thought trauma to left side of head 24 years ago caused beginning of deafness	
Nystagmus on irrigating both ears; dizziness and noises greater on left	Feeling of pressure in lower front part of chest in attacks; tight feeling in occiput; patient felt "cold all over"; tendency to fall to left, but did not fall; flashes of light to right after attacks, also dark spots in fields of vision and severe headache; onset of tinnitus and deafness 18 years before operation; dizzy spells and vomiting for 1 year which disappeared until 1 year before operation	.....	Avertin	7 days	No	Never changed	.....	
Not tested	Feeling of pressure in anterior left lower part of chest; sensation of fulness in left side of neck persisted after attacks; throbbing in ear after attacks; returning facial function began in 27 days and improved steadily and rapidly	.....	Avertin	No	No	No	.....	Facial paralysis developed 4 days after operation; cleared in 6 weeks
No response to caloric test on left; rotation test showed some function	Frontal headaches and patient staggered to right in attacks; rings in fields of vision; perhaps momentary loss of consciousness; attacks of migraine became milder as Ménière's disease developed; weakness after attacks; queer feeling in head, usually a premonitory symptom which developed shortly before operation; no increased loss of hearing within 18 months	Exploration for cerebellar tumor	Ether	4 days	No	No	Patient had migraine	1½ years before operation, internal auditory artery clipped; no effect
Not tested	Positive Babinski reflex and ankle clonus on left; constant sensation of dizziness between attacks; first attack lasted 3 weeks; began when patient raised his head in bed; dizziness increased by moving head; no nausea	.....	Local	2 years	No	Never changed	.....	Because of severe postoperative dizziness, wound opened and facial nerve injured; corrected by spinofacial anastomosis
Normal	Sensation of turning to right.....	.....	Local	Several months	No	Less than before	Patient has had operation on thyroid for exophthalmic goiter	
Normal,	Uncertain balance between attacks; hearing improved and diminished at times; patient had attacks of dizziness and unilateral deafness 5 years after tonsillectomy, disappeared until 10 days before operation when returned with great severity; stuffy feeling in ear and marked increase in deafness; tinnitus continued through 5 years' free interval; marked decline in hearing in interval	Tonsillectomy	Avertin	Yes	Several months	No	Influenza preceded first attack	
No response to irrigation on left	Intense desire to urinate and defecate; had to lie on bedpan during attacks; staggered between attacks; sense of pressure and numbness in head; for years unable to lie on left side; movement to left intensified dizziness	Gallbladder and appendix removed; tonsillectomy	Ether	No	No	Yes	.....	Tendency to stagger disappeared since operation
No response to caloric or rotation test on right, and only slight response on left	Staggered between attacks for 8 months preceding operation; following attacks severe frontal headache for from 3 to 6 hours; shortly before operation had tinnitus in opposite ear for a week, which disappeared; before operation sudden turning of head induced dizziness; attacks began with tight feeling in ear and recurred frequently	.....	Ether	A few weeks	No	No	.....	Facial paralysis from injury to nerve; corrected by spinofacial anastomosis
Normal	Loud cracks in ear at times; tinnitus constant; noises made attacks worse; attacks began with feeling of warmth between shoulders and over back of neck, then dizziness, nausea and vomiting; only left side of head felt dizzy; nystagmus with attacks; formerly sudden severe attacks of pain in ear and temple	.....	Rectal, ether	No	....	.....	Patient said deafness in left ear followed influenza in 1918	
No response to caloric test on left	Diplopia on one occasion, perhaps unconscious for from 1 to 2 seconds; constant frontal and occipital headaches for 4 months; occipital headache worse on left; 3 months before operation a feeling of numbness in forehead and nose, on both sides, but none since	Tonsillectomy and and appendectomy	Rectal, ether	Slight	10 days	No	.....	
More intense and longer nystagmus on irrigation of left ear	Cold sweat in attacks; patient staggered for a day or two after; slight nystagmus	.....	Avertin	5 days	No	No, but present for 1 year	.....	
Normal	Obliged to have head up in attacks to get most relief; tinnitus for 40 years, associated with onset of deafness, slight constant, slowly progressive; attacks came with lightning suddenness; sensation of drifting away in attacks	.....	Avertin	Slightly for 7 days	No	No	Deafness in this ear 40 years before operation after a cold	
Normal	Drawing sensation in back of head.....	.....	Avertin	10 days	No	Never changed	.....	
Normal but	Obliged to lie on back in attacks; occasional attacks when	....						

	1928	M	Right	Dizziness and deafness	3 years	3 years	3 years	None	3 years; none in attacks shortly before operation	3 years; none in attacks shortly before operation	About 1 per month	Up to 3 days	2 months	Increased before and during attack; less after	Objects turn	Only slig hearing remains
	J. R. H. 1929	43 M	Left	Deafness	2 years	2 years	10 years	Buzzing [illegible] 2 years	2 years	2 years; absent shortly before operation	Several every week; increased	Up to several hours; average, 1 hour	3 months	.....	Objects turn to right	Subtotal
16	E. R. 1929	41 M	Left	Dizziness and deafness	1½ years	1½ years	1½ years	6 months	None	In one attack only	1 per week	Up to 2 hours	9 months	Increased during attack	Objects turn clockwise	Subtotal
17	C. H. 1930	57 M	Left	Deafness	3 years	3 years	3 years	Constant for 3 years	3 years	3 years	1 per month	From 3 to 6 hours	6 months	Increased during attack	Objects rotate	Moderate also son loss in other ea
18	L. F. B. 1930	60 M	Right	Tinnitus	2 years	2 years	40 years	40 years	Only in most severe attacks	Only in most severe attacks	1 per week; maximum, 3 per day	3 hours	.....	Not at all	Objects rise up in front of patient	Few tone remain
19	M. T. 1930	38 F	Right	Dizziness	20 years	20 years	10 years	10 years	20 years	20 years	1 or 2 per week; formerly 1 per year	From 15 minutes to ½ day; average 2 hours	1 year	Increased during attack	Worse when lying, especially on back	Marked; loss for tones in other ea
20	A. L. 1930	45 M	Right	Dizziness	30 years	30 years	10 years; now bilateral	10 years; now bilateral	30 years	30 years	2 or 3 per month	From 1 to 24 hours	12 years	Increased before attack	Objects rotate from right to left	Marked; ilar in both ear
21	C. Z. 1931	53 F	Left	Tinnitus	14 years	2 years	14 years	14 years; bilateral	2 years	2 years	Numerous	Several hours	.....	Increased before attack	Objects whirl	Marked
22	J. A. B. 1931	70 M	Left	Deafness	9 months	9 months	30 years	Several	9 months	9 months	2 or 3 per day	From 10 to 15 minutes	A few weeks	Did not change	Objects "jumbled up"	Subtotal
23	W. F. F. 1931	60 M	Right	Deafness	8 months	8 months	5 years; sudden loss over night	5 years	8 months	8 months	2 or 3 per week	3 hours	1 month	Did not change	Objects whirl	Marked l eral, mu more on
24	N. M. 1931	69 F	Right	Tinnitus and deafness	4 years	4 years	5 years	5 years; some in opposite ear	4 years	4 years	.....	From 3 to 4 hours	2 years	.....	Objects whirl	.....
25	E. M. 1932	61 F	Left	Tinnitus and deafness	2 years	22 months	2 years	2 years	22 months	22 months	2 or 3 per month; at time of operation, 3 or 4 per day lasting from 15 to 20 minutes each	2 days for severe attacks; from 15 to 20 minutes for mild attacks	2 months	Increased before and during attack; less after	.....	Hearing almost g
26	W. W. 1932	42 M	Left	Deafness	A little less than 3 years	A little less than 3 years	3 years	A little less than 3 years	A little less than 3 years	A little less than 3 years	Every 2 or 3 weeks	From a few minutes to a week	A few months	Increased during attack	Objects revolve from right to left	Subtotal
27	F. K. 1932	49 M	Left	Deafness	17 months	17 months	18 months	Constant roaring for 17 months	17 months	17 months	2 per week	From 1 to 2 hours	6 months	No change	Objects rotate	Subtotal; some los on right
28	J. B. B. 1927	33 M	Left	Dizziness, deafness and tinnitus	2½ years	2½ years	2½ years	2½ years	None	None	1 per week	Up to 1 day	6 months	.....	Objects rotate	Subtotal
29	R. W. 1931	56 F	Left	Dizziness	11 years	11 years	6 years	Shortly after onset; constant	11 years	11 years	1 in 2 months but severe	From 2 hours to 3 weeks	10 years	Increased before and during attack	Objects whirl in every direction	50% loss
30	P. C. 1932	38 M	Left	Tinnitus	14 months	14 months	15 months	15 months	15 months	15 months	2 or 3 minor attacks per day	Up to 3 hours	10 months	No change	Objects rotate in every direction	Subtotal
31	C. L. R. 1933	26 M	Right	Tinnitus and deafness	13 years	13 years; 6 weeks	13 years; 6 weeks	13 years; formerly in left ear also	13 years	13 years	2 or 3 per week	Up to 2 days	12 years	Increased during attack	Objects turn to right	Subtotal; some los other ea
32	J. E. B. 1932	49 M	Right	Throbbing, full feeling in ear	1 month	1 month	Not known	1 month; occasionally in left ear	1 month	1 month	1 attack only	3 days	1 month	Increased during attack	Objects turn	Subtotal
33	F. S. 1933	51 M	Right	Deafness (f); dizziness	9 months	9 months	27 years	9 months	9 months	9 months	Every 2 or 3 days	From 10 minutes to 2 hours	1 month; 27 years	No change	Objects appear to fall	Subtotal; some los in other ear also
34	L. S. 1933	54 M	Left	Tinnitus	5 years	5 years	1 year, but old deafness in both ears; more on right	6 years	At times	At times	1 or 2 per week	From ½ hour to 24 hours	1 month	Increased	Objects turn in every direction; patient feels drunk	Subtotal both ear 55% loss right; 50 loss on l
35	R. L. P. 1933	55 M	Left	Deafness	1½ years	1 year	5 years	1 year; slight	Yes	Yes	2 or 3 per week; almost daily at time of operation	From 1 hour to 24 hours	1 month	No	Objects turn in every direction	50% loss; some los high ton on right
36	H. R. 1933	55 M	Right	Deafness	1 year	6 months	1 year	Slight; 7 months; antedated dizziness	Yes	Yes	About 20 attacks; 1 a week at time of operation	From 5 minutes to 4 hours	1 month	No	Objects whirl	12% loss right; 5 loss on l
37	C. N.	48	Right	Tinnitus	2 months	2 months	6 months	2 years	Yes	Yes	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

y slight aring nains	No response to caloric or rotation test on right, and only slight response on left	Staggered between attacks for 8 months preceding operation; following attacks severe frontal headache for from 3 to 6 hours; shortly before operation had tinnitus in opposite ear for a week, which disappeared; before operation sudden turning of head induced dizziness; attacks began with tight feeling in ear and recurred frequently	.....	Ether	A few weeks	No	No	.....	Facial paralysis from injury to nerve; corrected by spinofacial anastomosis
total	Normal	Loud cracks in ear at times; tinnitus constant; noises made attacks worse; attacks began with feeling of warmth between shoulders and over back of neck, then dizziness, nausea and vomiting; only left side of head felt dizzy; nystagmus with attacks; formerly sudden severe attacks of pain in ear and temple	.....	Rectal, ether	No	....	.....	Patient said deafness in left ear followed influenza in 1918	
total	No response to caloric test on left	Diplopia on one occasion, perhaps unconscious for from 1 to 2 seconds; constant frontal and occipital headaches for 4 months; occipital headache worse on left; 3 months before operation a feeling of numbness in forehead and nose, on both sides, but none since	Tonsillectomy and and appendectomy	Rectal, ether	Slight	10 days	No	.....	
derate; so some s in her ear	More intense and longer nystagmus on irrigation of left ear	Cold sweat in attacks; patient staggered for a day or two after; slight nystagmus	.....	Avertin	5 days	No	No, but present for 1 year	.....	
v tones main	Normal	Obliged to have head up in attacks to get most relief; tinnitus for 40 years, associated with onset of deafness, slight constant, slowly progressive; attacks came with lightning suddenness; sensation of drifting away in attacks	.....	Avertin	Slightly for 7 days	No	No	Deafness in this ear 40 years before operation after a cold	
rked; also s for high es in her ear	Normal	Drawing sensation in back of head.....	.....	Avertin	10 days	No	Never changed	.....	
rked; similar in th ears	Normal but absent on left	Obliged to lie on back in attack; occasional attacks when 12 years old, a single attack when 25 then no more until 38 (7 years before operation); only nocturnal attacks until 6 years before operation; bilateral deafness and tinnitus about equal on two sides; right side chosen for operation because of queer sensation in right ear in attacks; normal vestibular response on this side, absent on other	.....	Avertin	Severe for 3 weeks; gradually lessened	Still present 3 years later	No	.....	
rked	Normal	Staggered to left; occipital headache after attacks; positive Romberg sign; patient nervous; lost 80 pounds (36.3 Kg.) within year preceding operation; dazed feeling in head; tightness in chest; tingling sensation in left side of head for a year	.....	Avertin	Persisted for 6 months; mild	No	Never changed	.....	
total	Normal	.....	.....	Avertin	No	No	No	.....	Facial paralysis developed 1 week after operation; disappeared in 6 weeks
rked bilat-al, much re on right	Normal	Gradual loss of hearing in other ear for 7 years; no tinnitus in left ear; tinnitus developed with deafness; constant for several years, then intermittent	.....	Avertin	No	No, but balance affected	No	.....	Internal auditory artery indented nerve
.....	.....	Some loss of hearing and occasional tinnitus in left ear also; not always nausea and vomiting with attacks	.....	Avertin	Slight	1 day	.....	.....	
ring most gone	Absent on both right and left	Severe general headaches with attacks and after.....	.....	Local	10 days	No	Yes, but character changed	.....	
total	Delayed response on left	Some headache in left temporal region; staggered; dimness of vision in severe attacks; revolving objects absent in attacks shortly before operation; heart attacks longer and more frequent; vomiting relieved attacks	.....	Local	A few days	No	Occurs occasionally	.....	
total; me loss right	Impaired function on caloric test; rotation normal	Headache in left temporal region; patient saw double in attacks at times; thought deafness followed a cold; deafness preceded attacks by 1 month	.....	Local	Yes	Still persisted 1 year later; slight	No	.....	Diplopia for a few days after operation
total	Absent on left	Headache on left side.....	Tonsillectomy; gallbladder removed	Ether	No	No	.....	.....	
loss	Not tested	During first 2 weeks had an attack daily; no more until 1 year before operation; six attacks within year preceding operation; one attack lasted for several weeks continuously	.....	Avertin	10 days	No	Never changed	.....	
total	Normal	Heavy full feeling on left side of head; little dizziness with first attack; tinnitus, nausea and finally vomiting; tinnitus and deafness 1 month before other symptoms; dizziness severe only after 3 months; many minor attacks and few severe major ones; for 10 months only minor attacks, then a major one	.....	Avertin	No	No	Still present	.....	
total; me loss in her ear	Diminished	Patient tended to fall to right; 13 years before operation, at the age of 13, had during 4 months several attacks of bilateral tinnitus, vertigo, nausea and vomiting, each attack lasting from 1 to 2 days; tinnitus persisted since, more in the right ear; no more attacks occurred until 6 weeks before operation, when tinnitus increased and deafness increased markedly; no dizziness at that time	.....	Avertin	10 days	.....	Yes	.....	Remarkable antecedent history doubtless related to present attacks; diplopia for 3 days after operation
total	Normal	Probably had migraine.....	.....	Avertin	10 days	.....	Yes	.....	
total; me loss other r also total in th ears; % loss on ht; 50% s on left	Diminished	Attacks occurring in sleep awakened patient; slight deafness in this ear for 27 years, but apparently no progression until after first attack	.....	Avertin	3 weeks	No	Yes	.....	Only vestibular division of nerve divided; hearing remained unchanged after operation
	Normal	Frontal headaches and sensations of floating antedated attacks of dizziness; lasted for from 2 to 3 hours; gradual loss of hearing in both ears for 20 years, more on right; this condition stationary for past 3 years; daughter and mother had similar story of early deafness; tinnitus synchronous with heart beat	.....	Avertin	Severe	Yes	No	No	Internal auditory artery dented under surface of nerve
loss; me loss for gh tones right	Slightly retarded	.....	.....	Avertin	Slight	2 weeks	No	No	Only vestibular nerve divided; it was distinct from cochlear nerve
loss on ht; 52% s on left	Normal	Unsteady on feet at times between attacks; completely disoriented during severe attacks; transient periods of diplopia	.....	Avertin	Slight, 1 day	No	No	No	

37	C. N. 1933	43 M	Right	Tinnitus (intermittent at first, then constant for a year)	2 months	2 months	6 months	2 years	Yes	Yes	Almost daily	From a few minutes to 2 hours	2 weeks	Changes in pitch	Objects whirl in every direction	3% loss left; 3 on rig
38	E. A. C. 1933	52 M	Left	Tinnitus and deafness	2 years	2 years	15 years	15 years	Yes	Yes	1 per month	Up to 3 hours	3 months	.....	Objects whirl to right	3% loss right; loss or
39	W. I. R. 1933	43 M	Left	Dizziness	20 years	20 years; attack 10 years later	8 months gradually progressive	Constant buzzing for 2 years	Yes	Yes	At first far apart; almost daily at time of operation	Up to 1 hour	10 years	No	Objects whirl	42% los left
40	R. C. 1933	40 F	Left	Tinnitus	16½ years	16½ years	12 years	17 years, then disappeared for a time; bilateral during 2 years before operation	Yes	Yes	1 per week	Several hours	3 years	No	Objects turn from right to left	60% los left; s loss or right
41	L. D. 1933	59 F	Left	Dizziness	12 years	12 years	6 years	4 years	Yes	Yes	1 or 2 per week	15 minutes	.....	.....	Objects turn to right	50% los left; 1 on rig
42	L. V. L. 1933	44 F	Left	Tinnitus	7 months	7 months	6 months	Constant for 6 months; tinnitus for 4 days in same ear 7 years before operation	Yes	Yes	2 or 3 per week	2 hours	2 months	Increased	Objects whirl	18% los
24	N. M. 1931	60 F	Right	Tinnitus and deafness	4 years	4 years	5 years	5 years; some in opposite ear	4 years	4 years	.....	From 3 to 4 hours	2 years	.....	Objects whirl	.....
25	E. M. 1932	61 F	Left	Tinnitus and deafness	2 years	22 months	2 years	2 years	22 months	22 months	2 or 3 per month; at time of operation, 3 or 4 per day lasting from 15 to 20 minutes each	2 days for severe attacks; from 15 to 20 minutes for mild attacks	2 months	Increased before and during attack; less after	.....	Hearing almost
26	W. W. 1932	42 M	Left	Deafness	A little less than 3 years	A little less than 3 years	3 years	A little less than 3 years	A little less than 3 years	A little less than 3 years	Every 2 or 3 weeks	From a few minutes to a week	A few months	Increased during attack	Objects revolve from right to left	Subtot
27	F. K. 1932	49 M	Left	Deafness	17 months	17 months	18 months	Constant roaring for 17 months	17 months	17 months	2 per week	From 1 to 2 hours	6 months	No change	Objects rotate	Subtot some l on rig
28	J. B. B. 1927	33 M	Left	Dizziness, deafness and tinnitus	2½ years	2½ years	2½ years	2½ years	None	None	1 per week	Up to 1 day	6 months	.....	Objects rotate	Subtot
29	R. W. 1931	56 F	Left	Dizziness	11 years	11 years	6 years	Shortly after onset; constant	11 years	11 years	1 in 2 months but severe	From 2 hours to 3 weeks	10 years	Increased before and during attack	Objects whirl in every direction	50% los
30	P. C. 1932	38 M	Left	Tinnitus	14 months	14 months	15 months	15 months	15 months	15 months	2 or 3 minor attacks per day	Up to 3 hours	10 months	No change	Objects rotate in every direction	Subtot
31	C. L. R. 1933	26 M	Right	Tinnitus and deafness	13 years	13 years; 6 weeks	13 years; 6 weeks	13 years; formerly in left ear also	13 years	13 years	2 or 3 per week	Up to 2 days	12 years	Increased during attack	Objects turn to right	Subtot some l other
32	J. E. B. 1932	49 M	Right	Throbbing, full feeling in ear	1 month	1 month	Not known	1 month; occasionally in left ear	1 month	1 month	1 attack only	3 days	1 month	Increased during attack	Objects turn	Subtot
33	F. S. 1933	51 M	Right	Deafness (?); dizziness	9 months	9 months	27 years	9 months	9 months	9 months	Every 2 or 3 days	From 10 minutes to 2 hours	1 month; 27 years	No change	Objects appear to fall	Subtot some l in oth ear als
34	L. S. 1933	54 M	Left	Tinnitus	5 years	5 years	1 year, but old deafness in both ears; more on right	6 years	At times	At times	1 or 2 per week	From ½ hour to 24 hours	1 month	Increased	Objects turn in every direction; patient feels drunk	Subtot both e 55% lo right; loss or
35	R. L. P. 1933	55 M	Left	Deafness	1½ years	1 year	5 years	1 year; slight	Yes	Yes	2 or 3 per week; almost daily at time of operation	From 1 hour to 24 hours	1 month	No	Objects turn in every direction	50% los some l high t on rig
36	H. R. 1933	55 M	Right	Deafness	1 year	6 months	1 year	Slight; 7 months; antedated dizziness	Yes	Yes	About 20 attacks; 1 a week at time of operation	From 5 minutes to 4 hours	1 month	No	Objects whirl	12% los right; loss or

* In no case did the attacks persist after section of the nerve. The answer to the question, "Does anything precipitate attacks?" was negative in all but a few instances. Two patients thought that change of posture might be responsible. One believed that lying on the left side, and another that stooping and lying on the left side might be a factor. Movement of the head was given as a possible cause in one case, and turning the head suddenly at times in another. Most of the patients reported that section of

more on right		several years, then intermittent				affected			
.....	.....	Some loss of hearing and occasional tinnitus in left ear also; not always nausea and vomiting with attacks	.....	Avertin	Slight	1 day	.....	.....	
earing almost gone	Absent on both right and left	Severe general headaches with attacks and after.....	.....	Local	10 days	No	Yes, but character changed	.....	
ubtotal	Delayed response on left	Some headache in left temporal region; staggered; dimness of vision in severe attacks; revolving objects absent in attacks shortly before operation; heart attacks longer and more frequent; vomiting relieved attacks	.....	Local	A few days	No	Occurs occasionally	.....	
ubtotal; some loss in right	Impaired function on caloric test; rotation normal	Headache in left temporal region; patient saw double in attacks at times; thought deafness followed a cold; deafness preceded attacks by 1 month	.....	Local	Yes	Still persisted 1 year later; slight	No	.....	Diplopia for a few days after operation
ubtotal	Absent on left	Headache on left side.....	Tonsillectomy; gallbladder removed	Ether	No	No	.....	.....	
% loss	Not tested	During first 2 weeks had an attack daily; no more until 1 year before operation; six attacks within year preceding operation; one attack lasted for several weeks continuously	.....	Avertin	10 days	No	Never changed	.....	
ubtotal	Normal	Heavy full feeling on left side of head; little dizziness with first attack; tinnitus, nausea and finally vomiting; tinnitus and deafness 1 month before other symptoms; dizziness severe only after 3 months; many minor attacks and few severe major ones; for 10 months only minor attacks, then a major one	.....	Avertin	No	No	Still present	.....	
ubtotal; some loss in other ear	Diminished	Patient tended to fall to right; 13 years before operation, at the age of 18, had during 4 months several attacks of bilateral tinnitus, vertigo, nausea and vomiting, each attack lasting from 1 to 2 days; tinnitus persisted since, more in the right ear; no more attacks occurred until 6 weeks before operation, when tinnitus increased and deafness increased markedly; no dizziness at that time	.....	Avertin	10 days	.....	Yes	.....	Remarkable antecedent history doubtless related to present attacks; diplopia for 3 days after operation
ubtotal	Normal	Probably had migraine.....	.....	Avertin	10 days	.....	Yes	.....	
ubtotal; some loss in other ear also	Diminished	Attacks occurring in sleep awakened patient; slight deafness in this ear for 27 years, but apparently no progression until after first attack	.....	Avertin	3 weeks	No	Yes	.....	Only vestibular division of nerve divided; hearing remained unchanged after operation
ubtotal in both ears; 5% loss on right; 50% loss on left	Normal	Frontal headaches and sensations of floating antedated attacks of dizziness; lasted for from 2 to 3 hours; gradual loss of hearing in both ears for 20 years, more on right; this condition stationary for past 3 years; daughter and mother had similar story of early deafness; tinnitus synchronous with heart beat	.....	Avertin	Severe	Yes	No	No	Internal auditory artery dented under surface of nerve
% loss; some loss for high tones on right	Slightly retarded	.....	.....	Avertin	Slight	2 weeks	No	No	Only vestibular nerve divided; it was distinct from cochlear nerve
% loss on right; 52% loss on left	Normal	Unsteady on feet at times between attacks; completely disoriented during severe attacks; transient periods of diplopia	.....	Avertin	Slight, 1 day	No	No	No	
% loss on left; 37% loss on right	Normal	Transient periods of diplopia; at times slight headache on right side after attacks; previous double thyroidectomy (5 years before operation)	.....	Avertin	Slight, 1 week	No	No	No	Diplopia for 5 days after operation
% loss on right; 58% loss on left	Slightly diminished	Attacks became more severe; buzzing in left ear began about time of return of otitis media (15 years before operation), which was present in childhood; slight deafness noted at that time; neither symptom disturbing until 2 years before operation	.....	Avertin	Slight, 10 days	No	No	No	Facial paralysis developed after patient left the hospital (11th day after operation); began to return within 10 days
% loss on left	Normal	Patient staggered to right.....	.....	Avertin	Slight, 5 days	No	No	No	Nerve lifted by arterial loop
% loss on left; some loss on right	Absent on left; diminished on right	Some roaring and deafness in right ear for past 2 years; patient constantly dizzy for 1 year; no attacks since	.....	Avertin	Severe	Present 6 months later, but diminishing	Yes	No	
% loss on left; 15% loss on right	Diminished	Intermittent sharp pains in ear (left); loss of consciousness in attacks	Mastoidectomy; all teeth removed; ear drum punctured	Avertin	Yes	No	No	No	
% loss	Normal	Diplopia in attacks; patient staggered.....	Fallopian tubes and ovaries removed	Avertin	Yes	No	Yes	No	Diplopia for several days after operation; partial division of the nerve (%); loss of high tones only

the nerve was not painful, although one noted sharp, repeated pain, one a sensation of whirling and nausea and one pain in the ear and dizziness. A history of infection of the ear on the side affected was given by only two patients, in one of whom aural infection had been present since childhood. The other had had aural infection, but the side was not known.

8 throbbing persisted for some time after an attack. When the tinnitus is altered before an attack, it is obviously an aura, indicating that the attack is about to begin.

Nausea and Vomiting.—Nausea was present during the attacks in all except three cases (10, 16 and 28). In several instances it was present in some attacks and absent in others. In case 4 nausea accompanied the dizzy spells during the first two years and was absent in all subsequent attacks (for three years). In case 5 it was present in the first attack only, and in case 8 in only a single seizure. In cases 6 and 34 it was present only occasionally.

Vomiting has much the same relationship to Ménière's disease as nausea, and usually the two occurred together or both were absent. However, this was not always true, as, for example, in case 8 there was no vomiting, and nausea occurred only once. In case 10 there was vomiting in each of the attacks, but no nausea. In case 16 vomiting occurred in one attack, but nausea in none. Usually the nausea, and particularly the vomiting, were extremely severe. In most instances the patient vomited several times before the attack was over. At times the vomiting was of the type described as projectile, but this was not usual. In one instance the nausea or vomiting continued for days, even after the dizziness had passed (case 4). Occasionally (in case 3 only) vomiting improved the dizziness.

FREQUENCY, DURATION AND CHARACTER OF ATTACKS

At first the attacks usually occurred at infrequent intervals, later increasing in frequency or intensity or in both. The longest free interval between attacks was sixteen years (case 7); in several cases it was ten years or more. Although the attacks were always severe, they were usually endured without thought of surgical relief until they occurred once or twice a month. In most instances they occurred once or twice a week; in case 39 they occurred almost daily.

The average duration of an attack was from three to four hours. The attacks might last for only a few minutes, but they frequently persisted for many hours or days. In case 1 there was almost no respite for more than a month. During this time nausea and vomiting were so extreme that nourishment could be obtained only by rectum or by means of hypodermoclysis. The duration of the attacks may vary greatly, in the same patient, i.e., from a minute to a week or more. However, there is a tendency to uniformity.

In the overwhelming percentage of cases there was no recognized stimulus to induce the attacks, which came on with great suddenness, not infrequently during sleep. In five cases the patients thought that change of posture, especially sudden turning of the head, might have

brought on some attacks, but even in these instances most seizures began without apparent cause. In three cases (13, 26 and 41) the attacks appeared to be induced by the fact that the patient lay on the left side (the lesions were left-sided). In one instance (case 13) the patient, fearful of attacks from this cause, had been unable to lie on the left side for years. In none of the remaining cases was any patient able to suggest even a possible accessory cause for the development of the seizures.

The attacks of dizziness usually developed with extreme suddenness and without warning, not unlike epileptic attacks. I have seen patients thrown to the floor with such violence that broken bones resulted. At times, however, there was a brief aura, such as fulness or tingling in the affected ear or nearby, increasing tinnitus, a feeling of warmth between the shoulders or on the back or the side of the neck, a sensation of floating, headache, a queer feeling in the head or nausea. Usually the patients were able and obliged to lie down; usually they lay flat on the back and closed the eyes to get a degree of comfort. In case 19, however, the attack was worse when the patient lay down, especially when she lay on the back, and in case 3 the most favored position was on either side. Rarely did other factors influence the attacks. Exceptions were case 15, in which noises made the attacks worse, and case 3, in which vomiting caused definite improvement.

CHARACTER OF DIZZINESS

The character of the dizziness was most variable. Always (with one exception—case 22) there was the sensation of objects moving, usually in a rotating fashion. In seventeen cases objects turned, but in no recognizable direction. In six cases they rotated in every direction, at times in one direction, again in another, and at other times in such utter confusion that direction of movement was not evident. In one case (18) the movement was always in an upward, and in another (33) in a downward, direction. In case 22 objects did not rotate but appeared to be jumbled. I was especially interested to learn the direction of rotation when it was well defined and constant. Curiously, in five cases the rotation of objects was toward the affected ear (cases 13, 26, 31, 40 and 41), and in an equal number of cases it was in the opposite direction, i.e., away from the affected ear (cases 11, 15, 16, 20 and 38). The direction of rotation is, therefore, valueless in determining the ear involved. Had the direction of rotation of objects shown a constant relationship to the nerve involved, it would have been an invaluable guide in determining the side of the lesion in cases of pseudo-Menière's disease and true Ménière's disease (perhaps the two are the same) before the onset of deafness.

DISTURBANCE OF FUNCTION OF THE AUDITORY NERVE

In every case unilateral deafness was present in some degree, usually to the point where the remaining hearing was of little, if any, practical value. It was my impression that the audiometer curves showed nothing characteristic, i.e., that a diagnosis of Ménière's disease could be

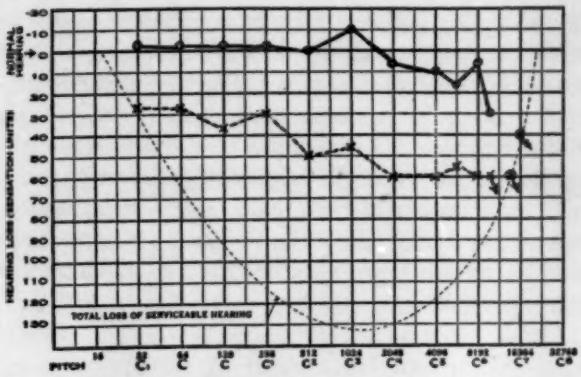

Fig. 1 (case 7).—Fairly typical audiometer curve in Ménière's disease. In this audiogram and in the following audiograms the unbroken line with the circles indicates the right side, and the broken line with the crosses, the left.

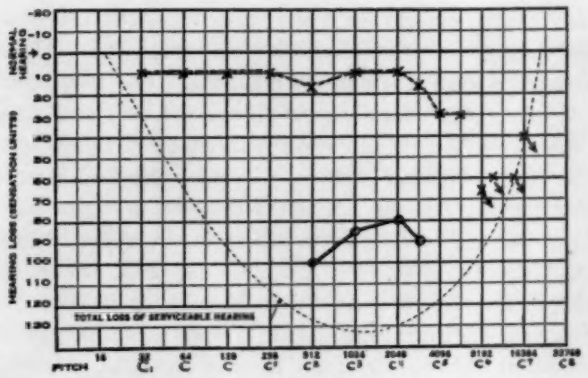

Fig. 2 (case 18).—Audiometer curve showing almost total loss of hearing in affected ear. Despite the extreme loss of hearing, there was no diminution in the frequency or severity of the attacks.

made from this source alone. However, Dr. Crowe, who has been especially interested in the study of the audiometer curves in Ménière's disease and in other lesions, such as acoutic tumors, middle ear deafness and deafness from other central causes, believes that the curves of Ménière's disease are distinctive, at least in many instances. In most of the cases of this series the hearing was charted as a low, flat, fairly

straight line, though with greater loss for high tones (fig. 1). The average loss of hearing in the affected ear of a patient with Ménière's disease will probably be found to be about 50 per cent, but it may be almost total (fig. 2).

The caloric (irrigation) test showed either diminished or absent function in 50 per cent of cases (eighteen of thirty-five patients examined). In eight (23 per cent) there was no response, and in ten (28 per cent) a diminished response, on irrigating the affected ear. But in one case (25) there was no response on irrigating either ear, and in another case (20) the response was normal in the affected ear and absent in the normal ear. I have little confidence in the caloric test in determining the degree of vestibular function. Indeed, the absence of demonstrable function by this test, when there is no reason to assume any vestibular abnormality (as in the two cases just mentioned), makes one somewhat skeptical as to whether evidence from this source indicates total loss of vestibular function in Ménière's disease.

OTHER SIGNS AND SYMPTOMS

In addition to the characteristic features of Ménière's disease, other subjective disturbances and, not infrequently, objective signs are superimposed. These may occur before, during and after the attack. The premonitory aurae have been mentioned. Many of the symptoms that occur during attacks persist for some time later, but perhaps more frequently they disappear with the attack. A great variety of disturbances was assembled from carefully taken histories. The most common complaints were queer sensations at, or about, the ear, and always on the same side. A queer feeling in the ear was noted in two cases, a tingling and tight feeling in the ear and (or) side of the neck in two cases, a warm flushed feeling in the ear in two cases, a tight feeling in the occiput in one case, a feeling of pressure in the chest in three cases, a sensation of "being cold all over" in one case, cold sweats in one case, a queer heavy feeling in the head but no pain or ache in two cases, a tingling sensation in the affected side of the head in one case and a drawing sensation in the back of the head in one case. Probably next in order of frequency were headaches; again, these might precede, accompany or (and) follow the attacks. In one case the headaches were general; in two they were frontal; in two occipital, and in two temporal; in four they covered the entire affected side of the head without further localization.

Dizziness persisted for varying periods of time after the attacks in four cases, and two patients had been constantly dizzy for many months. In two instances dizziness (not an attack) was precipitated by turning the head, reminding one of the transient dizziness of similar character that occurs in most instances following section of the nerve. During and

following attacks the patients staggered in sixteen cases. In eleven cases the staggering was not recognized as being to one side; in three instances it was toward the side of the lesion, and in three others, to the opposite side. In two cases a positive Romberg sign was present in the free interval between attacks when the patients entered the hospital.

Visual disturbances were not uncommon. Diplopia was present during and after attacks in five cases (16, 27, 36, 37 and 42)—again a reminder of the not infrequent diplopia occurring transiently following section of the auditory nerve (in four cases). Flashes of light to the opposite side occurred during, and persisted after, the attack in one case; dark spots or rings were noted in the two cases, and dimness of vision was noted in another case. Two of the patients with Ménière's disease were also victims of migraine. One of these stated that the migrainous attacks became milder as the number of dizzy spells increased. Whether there is any anatomic relationship between the two conditions can only be conjectured. Certainly, many of the superimposed symptoms of Ménière's disease suggest a spread of the stimulus to the autonomic nervous system, and it is my belief that migraine is a primary disturbance of this system.

Momentary loss of consciousness occurred in four cases, and in another case (18) there was the sensation of drifting away. Disorientation during the most severe attacks was claimed by one patient (case 36). A singular complaint (case 13) was an intense desire to urinate and defecate during the attacks; so severe was this urge that the patient was forced to lie on the bed pan during most of the seizure.

Excluding the findings referable to the auditory nerve, the only objective changes noted on examination of the patients were: lateral nystagmus (four cases), a positive Romberg sign (two cases), loss of the corneal reflex on the affected side (one case) and a positive Babinski sign and ankle clonus in the same patient, on the side of the lesion. Although objective findings are infrequent in Ménière's disease, it is important to recognize that they do not necessarily indicate an organic lesion of another type, such as a tumor or an aneurysm.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The diagnosis of Ménière's disease is rarely difficult. The essential features are recurring dizzy attacks, unilateral partial deafness and tinnitus in the affected ear. Until now a diagnosis of Ménière's disease would not have been made without all three features combined (there is one exception in this series, i. e., tinnitus was absent in case 14). The unilateral tinnitus and deafness differentiate the dizzy attacks of Ménière's disease from those due to cerebral arteriosclerosis and other known or unknown causes. Rarely do tumors in the cerebellopontile angle cause attacks of dizziness comparable to those of Ménière disease.

Usually, if dizziness occurs with tumors, it is only on turning the head and changing posture; it is of brief duration and does not assume the importance of well defined attacks. In a previous communication [1a] I reported similar attacks, with deafness and tinnitus resulting from an aneurysm of the basilar artery, but although several similar aneurysms

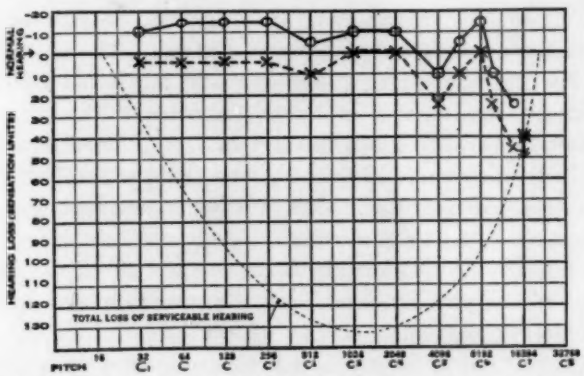

Fig. 3.—A diagnosis of pseudo-Ménière's disease was made from this audiogram, taken when the patient was first seen. At that time there was a slight drop in the hearing on the left side, but in the absence of tinnitus it was not definite enough to warrant division of the auditory nerve.

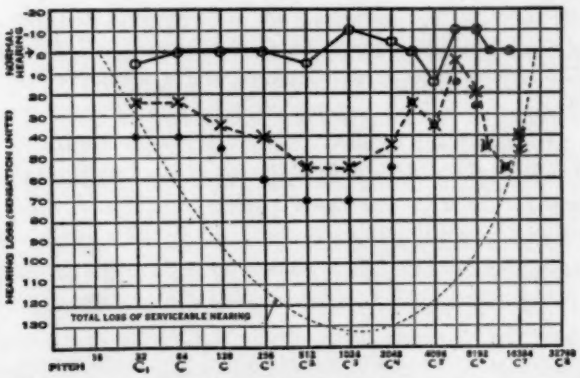

Fig. 4.—Audiogram showing the change in hearing of the same patient as in figure 3, after an interval of nearly two years. The diagnosis of Ménière's disease is now unmistakable. The heavy dots indicate thresholds for hearing on the left, determined with hearing on the right masked.

have since been observed, none has caused attacks of dizziness similar to those in Ménière's disease.

In nearly all cases of so-called acoustic tumors, the objective findings referable to the auditory nerve—in both the cochlear and the vestibular divisions—are similar to those in Ménière's disease, but I cannot recall

a single instance in which attacks of dizziness like those of Ménière's disease were also present. The differential diagnosis, therefore, between Ménière's disease and tumors in the cerebellopontile angle can be made, with rare exceptions (such as in the case of a basilar aneurysm), solely on the basis of the subjective symptoms.

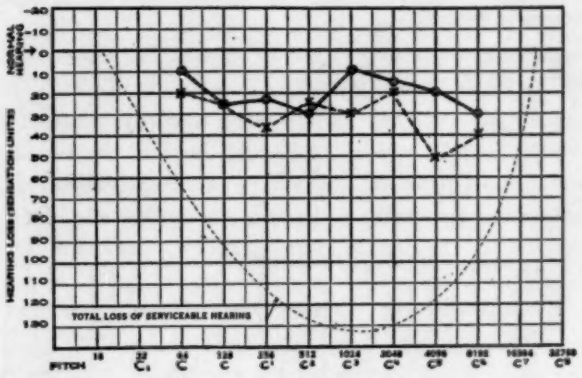

Fig. 5 (case 12).—Audiogram taken before sufficient evidence of the side affected had appeared.

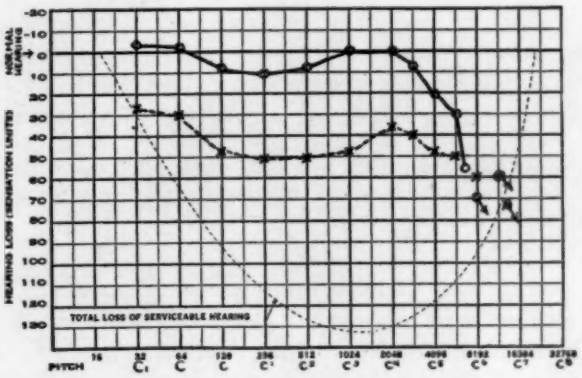

Fig. 6 (case 12).—Audiogram taken four and a half years after the first audiogram. Within this interval hearing on the left took a decided drop, leaving no doubt that the left ear was responsible for the attacks.

In attacks of pseudo-Ménière's disease the spells of dizziness are precisely like those of Ménière's disease, but tinnitus and deafness are absent. After studying table 3, one may wonder whether the former condition is not merely an early stage of the latter. It has been shown that in many cases the dizzy attacks antedated both tinnitus and deafness, even by a period of many years. Did these not, therefore, represent, in the beginning, pseudo-Ménière's disease, which later became well

defined Ménière's disease? A years ago, Dr. Crowe and I saw, in consultation, a patient of Dr. Barker's with pseudo-Ménière's disease and without tinnitus or unilateral deafness (fig. 3). Both deafness and tinnitus appeared later (fig. 4) making the diagnosis of Ménière's disease unequivocal. Another instance is case 12 (fig. 5 and 6). The importance of the diagnosis of Ménière's disease at such an early stage will be discussed later.

DIFFICULTY OF DETERMINING THE SIDE OF THE LESION

The number of patients (five) with marked bilateral deafness is noteworthy; this would appear to be well beyond the normal incidence. In three of these (cases 20, 23 and 34) the advanced deafness was almost equal in the two ears, and so far advanced was the loss that the

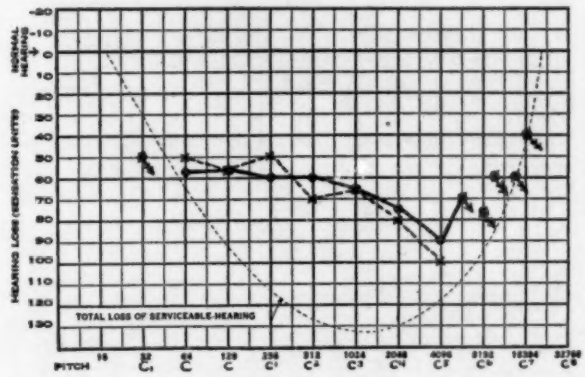

Fig. 7.—Audiogram of a patient who had almost equal loss of hearing in both ears. From the curve alone it would have been impossible to determine the side for sectioning of the auditory nerve.

remaining hearing was of little value, shouting, communication by lip reading or writing being necessary to get messages across. Indeed, under such circumstances, the choice of side on which to operate can be determined only with some uncertainty (fig. 7.) For example, in cases 23 and 34 the rather sudden and more recent loss of hearing was taken to indicate a newer process, probably related to Méninère's syndrome, but a choice would hardly be justifiable on the basis of this alone, since in many cases Ménière's syndrome has been engrafted on the side with long-standing deafness. In these cases the side of operation was determined more on the basis of the unilateral discomfort and roaring (tinnitus). In case 20 the choice was even more difficult, because the tinnitus was bilateral. Only the subjective symptom of a queer sensation in the right ear determined the side of operation. Against this was the objective evidence that the response to the caloric test was normal on

this side and absent on the other—a finding which caused much apprehension concerning the result. It has now been four years since operation in this case, and no attacks have developed.

IS MÉNIÈRE'S DISEASE EVER BILATERAL?

Whether Ménière's disease can occur bilaterally, as in trigeminal neuralgia (in which the condition is bilateral in about 2 per cent of all cases), cannot be definitely answered. One may well ask whether in all the cases with bilateral deafness and, perhaps, in those with bilateral tinnitus the condition is not of the bilateral type. But if it is bilateral, why should section of only one auditory nerve cure the disease, as it seemingly has done? One is led to wonder whether in this disease the central pathways for the functions of equilibration and hearing are not affected on both sides, in which event perhaps section of either vestibular branch would not be adequate; also, whether the transient postoperative dizziness that occurred in thirty-two of the forty-two cases studied (which will be discussed under the subject of postoperative sequelae) is not further evidence that the central pathways are bilaterally involved in the lesion (whatever it may be) causing Ménière's disease.

TREATMENT

Operation.—Until the advent of sectioning of the auditory nerve, there was no treatment, either curative or palliative, of Ménière's disease that is worthy of mention. Various medical and surgical efforts, usually based on erroneous conceptions of the nature of the disease, have been proposed, but have been futile. There is no point in dwelling on these. That Ménière's disease is now always curable by total section of the auditory nerve no longer admits of doubt. As has been previously noted, in not a single instance has there been an attack of dizziness subsequent to the operation in this series. Moreover, the operation is simple and safe. This operation was first performed by me on Jan. 11, 1927, i.e., more than seven years before the time of writing. The total number of such operations which I have performed to date is forty-two. Coleman and Lyerly [3] recently reported eleven cases in which this operation was performed and Cairns and Brain [4] reported four cases with identical results. The operative procedure has been described in one of my earlier publications.[1a] The approach to the nerve is not greatly different from that proposed in 1903 by Krause, of Berlin, for lesions (including tumors) of the cranial nerves. The only essential improvement—and

3. Coleman, C. C., and Lyerly, J. G.: Ménière's Disease: Diagnosis and Treatment, Tr. Am. Neurol. A. **58**:265, 1932.

4. Cairns, Hugh, and Brain, W. R.: Treatment by Division of the Eighth Nerve, Lancet **1**:946 (May 6) 1933.

it is very important—is evacuation of the cisterna magna before elevating the cerebellar hemisphere. Without this precaution, which supplies ample room in which to work, the cerebellum would frequently be severely damaged. It makes the approach to the cerebellopontile angle easy and safe. In his important pioneer publication, Krause reported section of the auditory nerve for persistent tinnitus, for which it was probably unsuccessful, but, curiously, it did not occur to him to use the same method in attacking Ménière's disease. Frazier (1907) and Ballance (1908) reported unsuccessful results in dividing the eighth nerve for tinnitus. Frazier sectioned the nerve for dizziness also, but without success, owing to the fact that the case was clearly not one of Ménière's disease.

The vertical limits of the incision (fig. 8) are parallel with the mastoid process; anteriorly, a horizontal limb curves mesially and downward, stopping about 5 cm. from the midline and 2 or 3 cm. above the

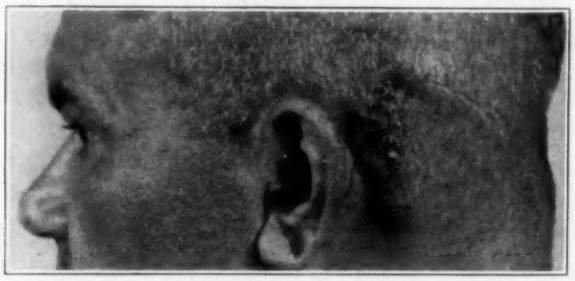

Fig. 8.—Scar showing site of operative approach in Ménière's disease.

base of the skull. Both ends of the curved incision are approximately on the same transverse plane, i.e., just above the tip of the mastoid. The trapezius muscle is divided transversely along its attachment and is stripped from the bone. Usually the occipital nerve and blood vessels are divided, but it is possible to avoid sacrificing them. A primary opening is made by a perforator, and the bony defect is progressively enlarged with rongeurs until approximately half of one side of the occipital bone has been removed. (fig. 9). Anteriorly the transverse sinus is reached, and laterally the mastoid cells are approached, but entry is carefully avoided. If by chance a cell is opened, a flap of dura is at once reflected over the opening and sutured. Mesially the bony defect ends at some distance (3 or 4 cm.) from the midline and at a somewhat shorter distance from the foramen magnum, the opening being extended farther in the latter direction in order to facilitate opening the cisterna magna. Immediately after incising the dura, the spatula is passed over the cerebellum and in the direction of the cisterna magna, the outer arachnoidal wall of which is pricked with forceps (fig. 8). A large

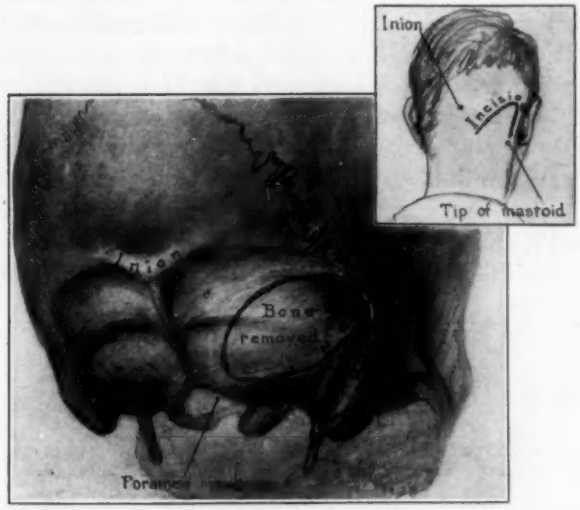

Fig. 9.—Sketch showing size and position of bony defect preparatory to exposure of the auditory nerve.

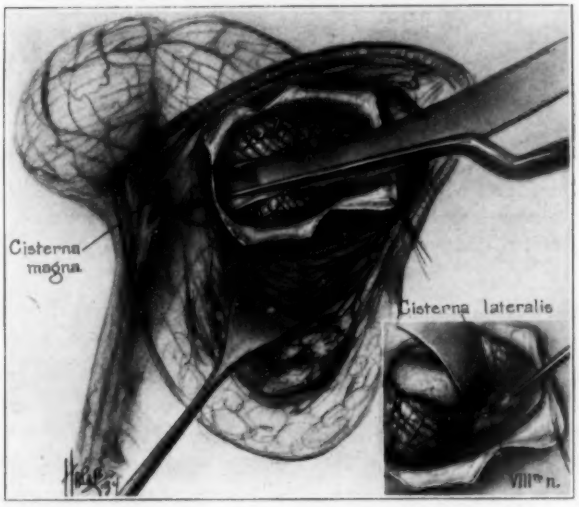

Fig. 10.—Sketch showing method of tapping, first, the cisterna magna, to obtain room in the posterior cranial fossa, and then the cisterna lateralis, to expose the eighth nerve.

quantity of cerebrospinal fluid is at once released and aspirated, after which the cerebellar lobe sinks well beneath the dura. Gentle elevation of the cerebellum then requires no pressure and consequently causes no trauma. The eighth nerve is the most superficial of the cranial nerves crossing the cerebellopontile angle and therefore promptly comes into view. The thin arachnoid sheath forming the outer wall of the cisterna lateralis is then punctured with forceps and gently stripped from the eighth nerve, which lies beneath and within the cistern (fig. 10). Occasionally thrombosis of a small artery or vein in the arachnoid must be produced and the vessel divided before the nerve can be safely exposed. Coagulation with the electrocautery, as suggested by Ward, makes this

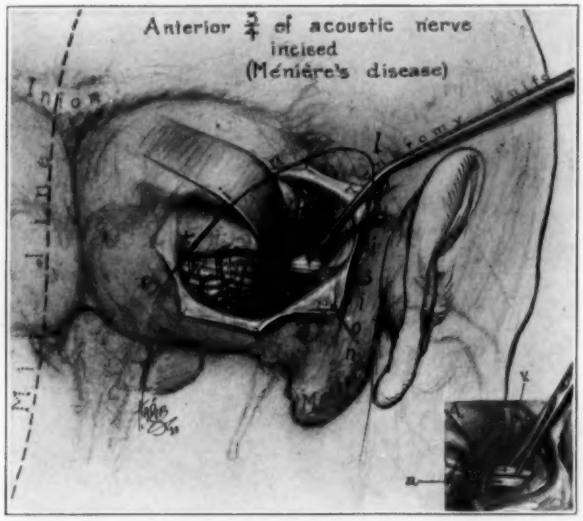

Fig. 11.—Sketch showing partial division of the auditory nerve, i.e., division of the vestibular branch, which comprises the anterior half of the nerve. This leaves the auditory branch intact or practically so.

procedure easy and safe. After aspirating the fluid in the cisterna lateralis (preferably by suction through pledgets of cotton), a blunt hook is passed beneath the auditory nerve in order to isolate carefully, and avoid injury to, the underlying facial nerve, which is nearly always completely hidden by the much larger eighth nerve.

Division of the auditory nerve may be made (1) by a sharp-angled knife, which is inserted beneath the nerve and drawn outward; (2) by small scissors; (3) by a small tenotomy knife, which is thrust through from without inward (fig. 11), (4) by pinching the nerve in repeated fractions with firm narrow-bladed forceps, or (5) by electrocoagulation. The choice of method is largely determined by fear of injury to the facial nerve, although this is now more a fear than an actual risk. A

small pledget of cotton can easily be inserted between the nerves as additional protection, but this is hardly necessary. For total division of a nerve, I prefer incision from without inward with a tenotomy knife or with tiny scissors, at the same time holding the nerve forward with a nerve hook. When there is not adequate room the latter precaution cannot be used. The electrocautery also works satisfactorily by coagulating and pinching successive fractions of the nerve. Instantaneous coagulation of small parts of the nerve softens it so that division by pinching is made easier. One must, of course, be certain that the facial nerve is well protected against injury during this procedure. Division of the nerve with the cutting current is even easier than this procedure and is not more dangerous. Another reason for preferring incision of the nerve from without inward is that any vessel lying beneath the nerve may be more easily seen and avoided. The internal auditory artery is usually visible and is easily separable from the nerve, but small branches may be obscured beneath. Although I have never injured a blood vessel, the fear of such an accident is always present.

In partial division of the nerve (division of the vestibular branch only), one must, of course, use much more care in order that the auditory division may not be injured. From other experiences in operating in the posterior cranial fossa, I know that the slightest trauma permanently destroys hearing. I prefer to use a small straight tenotomy or iridectomy knife, carefully drawing it from the anterior border of the nerve to its middle, or thrusting the tiny blade through the center of the nerve and drawing it anteriorly until half, or more, is severed. Pinching or the electrocautery may be equally effective, but I have been fearful of the effects of trauma or heat on the remaining cochlear fibers.

Closure.—The dura is always closed tightly. The importance of this cannot be overemphasized. In the first place, it is protection against a possible cerebrospinal fistula which might well be fatal, and in the second place, it prevents injury to the cerebellum, should postoperative vomiting, coughing or straining thrust the brain through the bony defect. Should the dura be shrunken from cauterization or some other cause, the dural defect is covered by a graft of fascia or muscle (from the trapezius) and sutured in place so carefully that a leak of cerebrospinal fluid cannot result.

Anesthetic.—Although the operation may easily be performed under local anesthesia and without pain—even section of the nerve is painless—I much prefer avertin anesthesia, unless there is some strong contraindication, and as yet there has been none. Avertin is far superior to ether because there is no swelling of the brain, with consequent reduction of room in which to work. And there is as much freedom from postoperative pneumonia with avertin anesthesia as with local anesthesia. If a choice must be made between ether anesthesia and local anesthesia, the latter is preferable.

POSTOPERATIVE SEQUELAE

Injury to the Facial Nerve.—In two of the earlier cases the facial nerve was severed. In one case this was due to a misconception concerning the nature of the immediate postoperative dizziness, which was then new to me. Fearing that the dizziness on turning following section of the auditory nerve indicated uncut fibers of the vestibular branch, I performed a second operation a few days later, and in the search through the angle the facial nerve was destroyed. Later the lost function was repaired by spinofacial anastomosis. In several of the earlier cases the operation was performed under local anesthesia in order that any stimulation of the facial nerve might be detected immediately by contractures of the facial muscle, but with increasing experience this precaution is no longer necessary. Moreover, under avertin anesthesia, facial contractions are perfectly evident when the nerve is touched.

Transient Bell's Palsy.—In three patients facial paralysis appeared late, i.e., several days after the operation, and cleared up spontaneously and completely. In case 8 loss of facial function first appeared on the fourth day and began to return within six weeks. In case 22 the paralysis began on the seventh day, and beginning function reappeared within six weeks. In case 38 paralysis developed two days after the patient left the hospital (on the eleventh day after operation) and began to return within ten days; later, within an additional three days, the patient thought that the function was about 60 per cent of normal; quickly thereafter it returned completely. The cause of the late motor paralysis could not be definitely established, although it would appear that this disorder was due to posttraumatic edema at the stump of the severed nerve. Doubtless the condition was essentially Bell's palsy, although in no instance was there associated pain, as so frequently obtains in this form of paralysis, perhaps owing to the fact that the intracranial part of the facial nerve is not tightly encased in a bony canal. When the first late palsy appeared, four years after the first operation for Ménière's disease, I could not escape the conviction that it was probably merely coincidental Bell's palsy and was unrelated to the operation, but the two additional cases make the incidence too high for such an explanation. Moreover, in each instance the paralysis was on the side on which the operation had been performed. Although it was unquestionably a sequel of the procedure, one wonders why it should develop so late. If it were due to traumatic edema the effect would be expected within from twenty-four to forty-eight hours. In an effort to prevent its development, the auditory nerve was sectioned at a greater distance from the porus acusticus internus (where the two nerves are in contact) and with a minimum of trauma, i.e., by avoiding, so far as possible, tugging at the nerve. It is interesting that in two instances the return of facial function began about six weeks after the palsy. Since this is

approximately the time known to be required for regeneration of the facial nerve—for example, in spinofacial anastomosis—it is perhaps fair to assume that regeneration may have occurred. However, in the remaining case (38) the return of function within ten days was much too quick for regeneration and suggests merely a rather prolonged physiologic block.

Diplopia.—In four cases diplopia followed operation and persisted for a few days. In no instance could examination disclose any extra-ocular weakness. Cairns and Brain stated that nearly all their patients had diplopia. Why there should be such a difference between their series and mine is difficult to understand. But why should diplopia ever follow the operation? It is not possible that one of the nerves supplying the extra-ocular muscles could be injured in any way at the time of operation; these nerves are too far afield; and even if such a possibility were conceded, there would be evidence of the defective function on objective examination. Possibly the diplopia is due to a slight muscular imbalance. This is also evident from its transient state.

Whatever the cause may be, it is doubtless the same as that of the diplopia that occurs at times in the course of the attacks of Ménière's disease. It is doubtless significant that, of the four patients of my series who had diplopia after the operation, three had had it during attacks before the operation (cases 27, 37 and 42). And of the five patients who had diplopia in their attacks (in one case [16] there had been diplopia in only one attack), three had it after the operation. The postoperative diplopia would, therefore, appear to be similar in origin to the diplopia occurring in the attacks; i.e., it was involved in the explosion of the various closely related functions which together made up the attacks.

Transient Nystagmus.—This also occurred with considerable frequency after operation; it usually disappeared within a few days, just as did the diplopia. It, too, occurred frequently during attacks and was a not infrequent finding between attacks.

Transient Vertigo.—Usually (in thirty-two of the forty-two cases studied) transient vertigo occurred immediately after operation. This must not be mistaken for an exacerbation of Ménière's disease, as was done in one of my earlier cases. The intensity and duration of the dizziness were variable. Dizziness might be absent (in ten cases) or mild, as in most instances; but occasionally it was severe. Usually it began on the day of the operation and reached its peak within forty-eight hours, after which it declined steadily, and in most instances it had practically disappeared at the end of a week or ten days. At first the dizziness might be continuous, but it was always intensified by turning the head or body or even the eyes. In this stage it was not unlike the dizziness of Ménière's disease, but it soon gave way to momentary

dizziness induced by turning the head only. At times nystagmus was also present, and nausea and vomiting were frequently associated during this period. The period of constant dizziness lasted as long as three weeks in one patient and two weeks in three others. The dizziness on turning the head occasionally persisted for a year or more; in one patient it persisted for three years. In one instance there was disturbance of balance on turning the head, but no dizziness. The patient was insistent about this differential point, which has also been brought out by others.

The explanation of this postoperative dizziness and its seemingly capricious character is by no means clear. Why should it be absent in some patients and mild or severe in others? And why should it usually disappear within a few days, but at other times endure for weeks or months? It hardly seems possible that some element involved in section of the nerve, such as the amount of trauma produced, would be a factor, for the result is essentially the same. To evaluate this element, the auditory nerve was cocainized (with 20 per cent cocaine) in a number of cases before division, but without any apparent difference in results. One may wonder whether the sudden loss of function resulting from severing the nerve to one vestibular apparatus and the assumption of the entire function of equilibrium by that on the other side was responsible. But such an explanation is quickly dispelled by the absence of dizziness when the eighth nerve is divided at operation in conditions other than Ménière's disease. I have just discharged from the hospital a young man who underwent an operation for total removal of a large encapsulated tumor (not an acoustic tumor) from the left cerebellopontile angle, necessarily with excision of the seventh and eight nerves. There was no dizziness after the operation; the degree of nystagmus was not increased, nor was there tinnitus or postoperative vomiting. Before the operation his hearing was shown by audiometer curves to have been normal in both ears; subsequent to the operation the left ear was, of course, totally deaf. In this connection, in cases of acoustic tumors with subtotal deafness of the same degree that occurs in Ménière's disease, at times with preservation of vestibular function, total division of the nerve, with resultant complete loss of all its functions, is not followed by postoperative dizziness of this type. One might also think of possible trauma to the cerebellum incident to its retraction as a possible cause, but the same elevation, though to a greater degree, is made during operations for trigeminal neuralgia; yet such dizziness does not follow operations of this kind, nor does it result from excision of the outer cap of the cerebellum as a preliminary step in the removal of tumors in the cerebellopontile angle.

The only explanation, therefore, for the postoperative dizziness, and this is only a partial explanation, is that the dizziness is in some way peculiar to Ménière's disease and is, therefore, referable to the underlying pathologic changes in this condition. One is prepared to believe from these conclusions that the underlying lesion is located somewhere in the central pathways responsible for dizziness. Certainly, if the lesion were in the semicircular canals, as is generally believed, section of the sensory root of the auditory nerve would at once cut off all stimuli that could induce dizziness from this source, just as division of the sensory root of the trigeminal nerve immediately and forever destroys all perception of sensation referable to this nerve in the face.

In an earlier publication,[1a] emphasis was placed on the significance in differential diagnosis of momentary dizziness induced by turning the head, as contrasted with the longer paroxysmal attacks observed in Ménière's disease. In the former type of dizziness the lesion was found to be within the central nervous system, i. e., the cerebellum, the brain stem, the third ventricle or even (though rarely) the cerebral hemispheres. But I have never known an attack of Ménière's disease to result from one of these lesions. After section of the eighth nerve in Ménière's disease the transient postoperative dizziness is precisely of the type to be expected following this procedure, i. e., that induced by turning the head, and since the end-organ (the semicircular canals) of that side no longer functions, the dizziness must be either independent of the semicircular canals of both sides or dependent on stimulation of the semicircular canals of the contralateral side only.

Is postoperative dizziness related to bilateral deafness? At one time I thought so. The two patients with perhaps the most intense postoperative dizziness had but little remaining hearing of practical value. On the other hand, the only other patient with essentially the same degree of bilateral deafness had no dizziness following the operation, and two other patients with slight loss of hearing in the contralateral ear had dizziness of nearly as severe a grade for two weeks after the operation. That the inherent nervousness of the patient accounts for a degree of this disorder is doubtless true, but that there is a fundamental organic cause for variations in its intensity cannot be doubted.

Although the prolongation at times of the second stage, i. e., of dizziness on turning the head, appears to have some relationship to the intensity of the immediate postoperative dizziness (cases 20 and 24), this is not always true. In two cases (10 and 40) the same type of dizziness on turning the head had been present continuously for many months before the operation. The postoperative dizziness was, therefore, doubtless but a continuation of the already existing condition, just

as is true with diplopia and, perhaps, tinnitus. It is my impression that the intensity and duration of both the postoperative continuous dizziness and that induced by turning the head are due to the original lesion, whatever it may be, in the vestibular-cochlear apparatus, and not to any factor (such as trauma or transfer of function to the other side) involved in the operative procedure. It is not improbable that many, at least, of the transient sequelae are indicative of the close anatomic relationship between the nervous pathways of the two sides and, therefore, of an actual or potential bilateral lesion of the vestibular-cochlear tracts.

Postoperative Tinnitus.—After operation the tinnitus disappeared (in twenty cases), remained unchanged (in seventeen cases) or was diminished (in two cases); it never increased. In one patient (case 17) the tinnitus persisted for a year and then vanished. In case 34 it disappeared after two weeks; in case 26 the noise recurred only occasionally, and in case 25 the character of the tinnitus was completely changed.

From the existence of extreme bilateral deafness in three cases and of preoperative continuous dizziness in two others, it is difficult to escape the conviction that the postoperative dizziness is dependent on bilateral involvement of the vestibular tracts. The reasons for the variability are not understoood. In a patient with tinnitus and total deafness, without dizziness, I divided the eighth nerve intracranially, but without appreciable change in the tinnitus. Other surgeons have had similar experiences. I have seen no report of a case in which section of the nerve has abolished the noises, although I have no doubt that in a percentage of cases, perhaps corresponding closely with the figures just given, the tinnitus may be cured by section of the nerve. The result could, however, scarcely be predicted. As has been previously noted, the early operations on the auditory nerve were for tinnitus. How far the nervous condition of the patient is responsible for the tinnitus, in many cases at least, I am unable to guess. It is evident that one can never promise a patient with Ménière's disease that relief or cure of tinnitus will follow. The chances of total disappearance are slightly better than 50 per cent, and those of improvement, if the condition persists, an additional 20 per cent. There is also some prospect that the tinnitus will eventually disappear, even though it may remain for a time after the operation.

ETIOLOGY AND PATHOLOGY

I have not as yet been able to find a single report of a necropsy in a case of Ménière's disease. Since the attacks do not cause death, the only postmortem material would be obtained incidentally. Now that patients will be operated on with increasing frequency, pathologic

material will doubtless appear from time to time, but it is extremely doubtful if any preoperative abnormality, excepting gross lesions, such as tumors, could be separated from the changes resulting from section of the nerve.

In an earlier publication [18] I expressed the belief that the lesion of Ménière's disease must be located in (or on) the eighth nerve and not in the end-organs. The reason for this assumption was that since both the hearing and the vestibular function were always affected, it was most unlikely that a lesion could be so diffuse as to involve both the semicircular canals and the cochlea. It therefore seemed highly improbable that the cause of Ménière's disease could be located in any position other than in the auditory nerve. From experience with many tumors located in all parts of the posterior cranial fossa, I have seen no instance in which a tumor within the brain stem or cerebellum has caused deafness and none in which attacks of dizziness like those of Ménière's disease have resulted. Tumors along the cerebellum and brain stem give rise only to the type of dizziness that is induced by change of position, and is never of the terrific protracted type characteristic of this condition. These observations led to the belief that the lesion is not in the brain stem or the pathways within the central nervous system. Moreover, the involvement of both vestibular and auditory tracts after their divergence in the brain stem would appear to be much less likely than their involvement in the auditory nerve. But there is no reason to assume that the lesion is single instead of diffuse.

On the other hand, tumors along the intracranial part of the auditory nerve apparently do not cause dizzy attacks like those of Ménière's disease, but rather dizziness induced by change of posture; and in these cases both the vestibular and the cochlear functions are altered during the progress of the tumor in all degrees between normal function and total abolition of function. In not one of the forty-two cases in this series in which operation was performed was a tumor found, nor was any gross lesion detected. The absence of gross changes is the more surprising because they are frequently found along the sensory root of the trigeminal nerve during operations for tic douloureux. Possibly the same gross lesions that induce pain do not provoke dizziness. Touching the pain fibers of the fifth nerve induces pain, but does not stimulate abnormal sensations of other types; nor does cutting the eighth nerve immediately induce dizziness, tinnitus or sounds in the ear. It is perhaps conceivable that the internal auditory artery, which is frequently large and which passes through the meatus with the nerve, may cause these disturbances, especially since Ménière's disease develops in adults; but a branch of the ophthalmic artery passing through the optic foramen apparently causes no analogous disturbances of function

of the optic nerve. In case 34 a loop of the internal auditory artery definitely elevated the nerve and in case 23 the nerve was indented by the artery. Before the present type of operation for Ménière's disease was attempted, the cerebellopontile angle was explored in one case, and the internal auditory artery was divided because of the suspicion that it might be the causative factor, but no improvement followed. Moreover, in operation for trigeminal neuralgia numerous vascular indentations of the auditory nerve have been seen in passing, but in these cases there has been no disturbance in function. One can only wonder why!

In its paroxysmal attacks, Ménière's disease closely resembles trigeminal or glossopharyngeal tic douloureux and also the convulsions of epilepsy. In all these conditions the patient is free from symptoms between the attacks. From studies of trigeminal neuralgia, I have concluded that the lesion causing the peculiar paroxysmal type of pain can be located only in the sensory root of the nerve and never in the ganglion or the peripheral branches. Similarly, the lesion causing convulsions (in epilepsy) can be located only in the cerebral hemispheres and never in the lower neuron. It would appear reasonable to expect that the attacks of Ménière's disease could be due only to a lesion in the sensory root, i. e., in the auditory nerve, and not in the end-organ (the semicircular canals).

Nothing positive has been disclosed concerning the etiology of Ménière's disease. As in trigeminal neuralgia, the disease attacks healthy adults. Only two patients of this series had had infection of the middle ear or mastoid—an incidence probably no greater than obtains in persons without Ménière's disease. In two other patients deafness was said to have followed a cold or influenza, but there was no discharge from the ear. On the other hand, among the great numbers of cases of infection of the middle ear or mastoid one does not see Ménière's disease as a sequel. Syphilis played no part in the condition in my series, and at a time when the rôle of focal infections with distant sequelae is stressed, one can say with certainty that there is no reason to suspect an etiologic relationship of this kind; the eradication of infected foci has not altered and cannot alter the course of the disease.

EFFECTS ON HEARING OF PARTIAL SECTION OF THE AUDITORY NERVE

It has already been stated that in three patients the cessation of attacks of dizziness after partial division of the auditory nerve was identical with that following total section; the elapsing time between the operation and relief was thirteen, seven and four months, respec-

tively. Occasionally (twice in this series) the cochlear and vestibular branches of the nerve were separate, but in all the other cases the intracranial portion of the eighth nerve showed no division between the two branches. Knowing that the vestibular part of the nerve is located anteriorly, one could only assume that the two branches were essentially of equal size and that division of the anterior half would

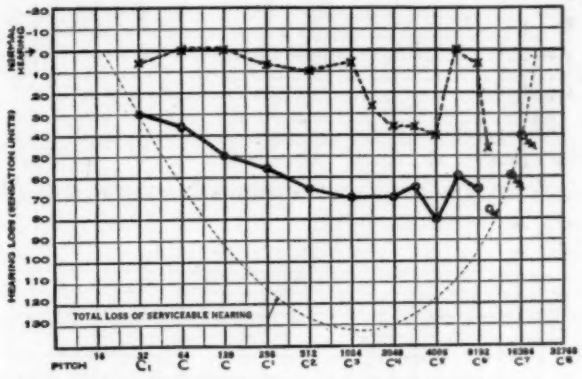

Fig. 12 (case 33).—Audiogram taken six days before operation in a patient in whom five eighths of the auditory nerve was divided.

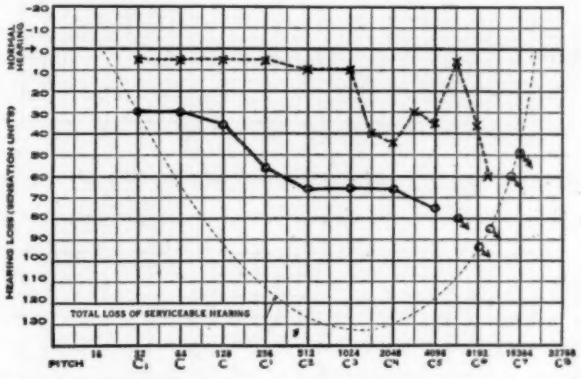

Fig. 13 (case 33).—Audiogram taken thirteen days after operation.

sever all the vestibular fibers. In order to be on the safe side, an additional cut into the posterior, or cochlear, half was thought advisable. The results in terms of hearing were most interesting. In the first case approximately five eighths of the entire nerve was divided. Assuming that the cochlear and vestibular nerves were of equal size, the result would be that 25 per cent of the cochlear fibers had been divided. No appreciable loss of hearing could be detected in the audiograms (figs. 12, 13 and 14), and after a lapse of thirteen months the audiometer

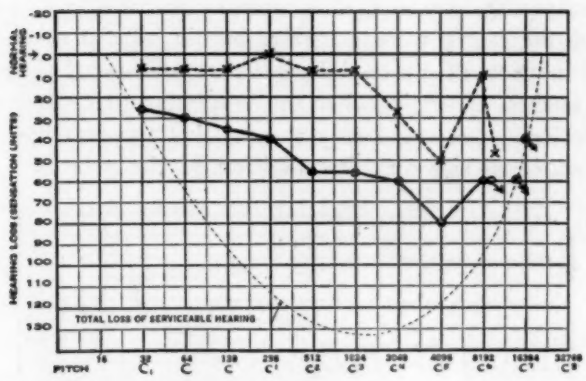

Fig. 14 (case 33).—Audiogram taken seven and a half months after operation.

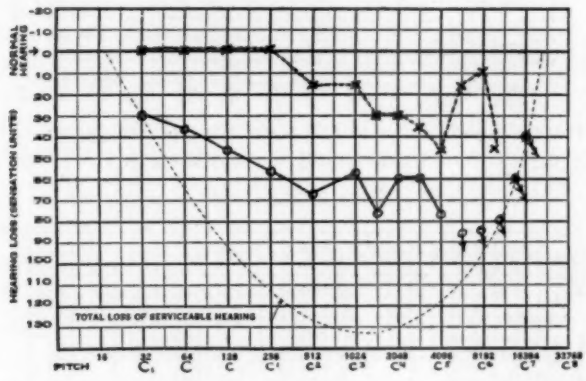

Fig. 15 (case 33).—Audiogram taken thirteen months after operation.

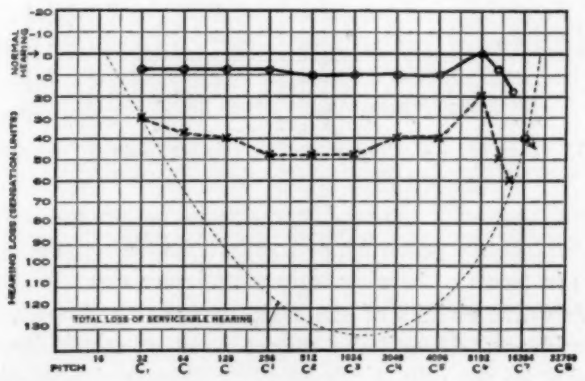

Fig. 16 (case 42).—Audiogram taken before operation in a patient in whom three fourths of the auditory nerve (one half of the cochlear branch) was divided.

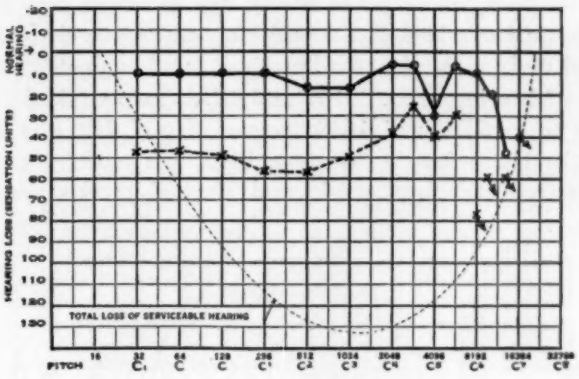

Fig. 17 (case 42).—Audiogram taken thirteen days after operation.

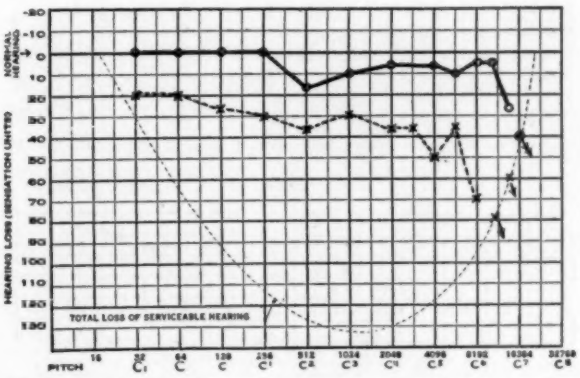

Fig. 18 (case 42).—Audiogram taken four months after operation.

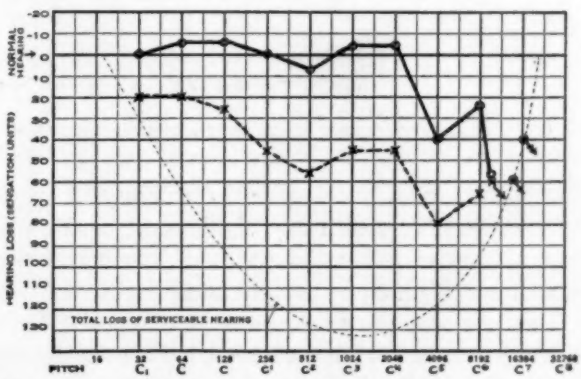

Fig. 19 (case 35).—Audiogram taken before operation in a patient in whom the isolated and independent vestibular branch of the nerve was divided.

curve was essentially the same as before the operation (fig. 15). In the third case at least one half of the cochlear branch was divided (assuming an equal division of the two branches), and again there was little, if any, loss of hearing (figs. 16, 17 and 18). Two weeks later hearing of the high tones 8192 and 16384 was equivocal, but at the last test (four months later) hearing of tone 8192 was again normal. Four months after operation an audiogram showed marked improvement

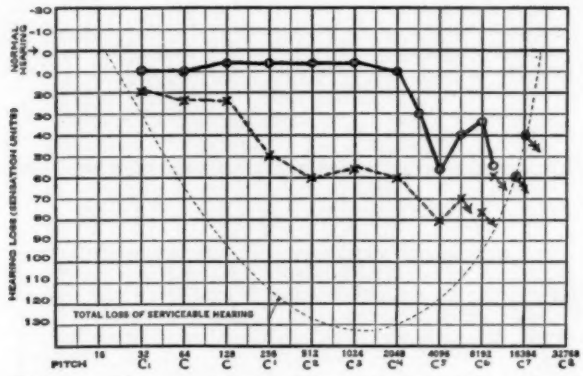

Fig. 20 (case 35).—Audiogram taken six days after operation.

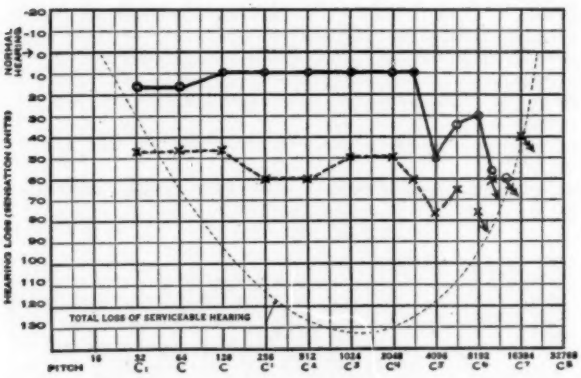

Fig. 21 (case 35).—Audiogram taken seven months after operation.

in the hearing of both ears, but despite the accuracy of the audiometer tests I am not inclined to attach any significance to the result, which may be only a normal variation. The second patient's vestibular nerve was well isolated and distinct, so that no additional loss of hearing was anticipated from the operation; nor did any appear. Seven months later, his hearing was just as it had been before the operation (figs. 19, 20 and 21). This patient was a railroad conductor, and the tenure of his position was dependent on the preservation of the hearing of the

affected ear. He was working for five months before the time of writing without any symptoms of his former disorder and without dizziness on turning the head.

It is, therefore, evident from two of these three cases that many auditory fibers in the sensory root can be sacrificed without loss of hearing. This result is in keeping with the absence of detectable function when parts of the sensory root of the trigeminal nerve, and, perhaps, even of the orbital nerve, are sectioned. It is interesting, too, that, after a year in the first case and after a lesser time in the two remaining cases, there was no further loss of hearing such as one might well have expected had the disease been untreated. However, the time has been too short to claim that this freedom from further auditory loss is permanent.

WHEN TO OPERATE

From these results, it is evident that one may now safely operate on patients with Ménière's disease in the early stages, when deafness is just beginning, and, perhaps, even on patients with pseudo-Ménière's disease, if there is any indication of the side involved. There is even the hope that bilateral division of the vestibular branch may be performed with impunity if unilateral section is not effective, in attacks of pseudo-Ménière's disease. Despite the oft-repeated statement of Charcot that the attacks stop when deafness on the affected side becomes total, there is rarely little hope of spontaneous cure. Only once have I seen a patient whose attacks ceased, and in this case deafness had not progressed. It is probable that in this instance the condition was merely in one of the infrequent long latent periods. It is clear that the degree of deafness can never be an index of the disturbance in the vestibular branch of the nerve, and since the attacks must be regarded as essentially vestibular, it is not improbable that a patient may be totally deaf and still have an intact and violently active vestibular tract. At best, therefore, deafness is only a fair guide in the late stages, as it is now known to be in the early stages, of the disease.

SUMMARY AND CONCLUSIONS

The signs and symptoms in forty-two cases of Ménière's disease are analyzed. In this small series the disease occurred nearly twice as frequently in males as in females and twice as frequently on the left side as on the right.

The symptom of onset may be dizziness, deafness or tinnitus, in about equal percentages.

The basic and accessory symptoms are enumerated and analyzed.

The diagnosis, differential diagnosis and character of Ménière's disease are discussed.

Ménière's disease may be cured by total section of the auditory nerve. There has been no known attack subsequent to the operation in any of the forty-two cases.

Partial section of the nerve, i. e., total division of the vestibular branch, was performed in three cases. The results have, so far, been identical, although only fourteen months and twelve months, respectively, have elapsed since the first two operations to the time of writing. Hearing is, of course, preserved by partial section, leaving no permanent after-effect of the operation.

The variable immediate but transient postoperative effects of the disease are enumerated.

The site of the lesion causing Ménière's disease is thought to be in the auditory nerve and not in the end-organ.

The hearing that was preserved by partial section of the nerve has so far remained intact. It is hoped, but not yet assured, that cure of the disease in the early stages may prevent the progressive loss of hearing that is inevitable if the condition is left untreated.

COLOR INDEX OF THE NASAL SEPTUM

A CRITICAL STUDY OF THE VASOMOTOR MECHANISM OF THE NOSE

L. B. BERNHEIMER, M.D.
CHICAGO

The etiology of nasal disease is complex, with important underlying constitutional factors remaining unrecognized or improperly interpreted. Jarvis [1] and his coworkers believed that an important constitutional factor in the production of infections and allergic nasal disease is an acid-base imbalance, that these imbalances can be rectified by diet, and that they can be recognized by the color of the nasal septal mucosa.

A recent report [2] gave the results of a critical study of the supposed value of the color of the nasal septum as an index of acid-base imbalance resulting from dietary insufficiencies. The following conclusions were drawn: 1. No correlation could be found between the color of the nasal mucosa and acid-base disturbances. 2. Diets high in foods producing alkaline or acid metabolic products failed to influence either the color of the septal mucosa or the symptoms of allergic or infectious nasal disease. Following this report, Jarvis [3] concluded that his theory of acid-base imbalance as a cause of allergic or infectious nasal disease was untenable, but he stated a belief that the excellent clinical results which he and his disciples claimed to have obtained through dietary treatment of these two types of nasal disease resulted from the effects of minerals, drugs, hormones and vitamins on the vasomotor mechanism of the nose. The purpose of the present communication is: (1) to discuss the vasomotor mechanism of the nose, (2) to present the results of a study of the effects of salts, organic compounds and hormones on the mechanism and (3) to present a tabulation of the color of the nasal septal mucosa found in persons with demonstrable vasomotor disturbances or vascular disease.

From the Department of Otolaryngology and the Cardiovascular Laboratory, Department of Physiology, Michael Reese Hospital.

1. Jarvis, D. C.: Group correspondence.

2. Bernheimer, L. B., and Cohn, D. J.: The Color of the Nasal Septum; Critical Study of Its Supposed Value as an Index for Dietary Therapy in Diseases of the Upper Respiratory Tract, J. A. M. A. **100**:1324 (April 29) 1933.

3. Jarvis,[1] p. 137.

THE VASOMOTOR NERVES OF THE NOSE AND THEIR PHYSIOLOGIC ACTIVITY

In considering the vasomotor mechanism of the nose the existence and physiologic activities of two antagonistic sets of nerve fibers must be considered, namely, the vasoconstrictor fibers which cause a contraction of the muscular coats of the arteries, and the vasodilator nerves which cause an increase in the size of the blood vessels, probably owing to a relaxation of the muscular coats.

The vasoconstrictor fibers belong to the sympathetic autonomic system and consist of preganglionic fibers arising in the central nervous system and postganglionic fibers arising from the cells of sympathetic ganglions. They constantly exhibit tonic activity, the impulses arising from the vasoconstrictor center in the medulla oblongata. This center is susceptible to reflex stimulation through sensory nerves, so that its action can be increased or decreased. The afferent nerve fibers which cause reflex vasoconstriction are called pressor fibers, and those which cause reflex vasodilatation are called depressor fibers. The pressor fibers are stimulated under some circumstances and the depressor fibers under others. For instance, ice applied to the nasal mucosa causes blanching and relieves congestion, the low temperature presumably stimulating sensory fibers in the mucous membrane and reflexly stimulating the vasoconstrictor center. Exposure to high temperature, on the other hand, causes a reddening of the nasal mucosa which is supposedly due to a reflex inhibition of the tone of the vasoconstrictor center. This center is also affected by psychic states, as is manifested by blushing resulting from embarrassment. The vasoconstrictor fibers of the nose, mouth and pharynx are derived mainly from the sympathetic autonomic system, arising from the spinal cord in the upper thoracic and eighth cervical nerves and proceeding by rami to the superior cervical ganglion where they are distributed as postganglionic fibers.

The vasodilators are not as powerful as the vasoconstrictors. In the case of the erectile tissue of the turbinates of the nose and the glands in the mucous membrane of the nose these fibers apparently are active only when reflexly stimulated. The exact nature of the action of the vasodilator fibers is not satisfactorily understood. No vasodilator center in the brain similar to the vasoconstrictor center has been conclusively demonstrated, although the presence of one has been suspected. The vasodilator fibers of the nose are supplied in part by the cervical sympathetic and in part by the seventh and ninth cranial nerves and the sympathetic ganglions with which they connect. The outflow of the seventh nerve passes to the sphenopalatine ganglion, whence it emerges as postganglionic fibers which accompany the superior maxillary

nerve, causing vasodilatation in the membrane of the nose, soft palate, tonsils, uvula, roof of the mouth and pharynx.

The caliber of blood vessels can be influenced otherwise than through the agency of vasoconstrictor and vasodilator fibers. Bayliss [4] has indicated that in addition to the nerve mechanism just described, chemical substances exert a regulatory influence. For example, lactic acid and carbon dioxide are produced in tissue during functional activity. These substances have been found to cause vasodilatation, thus providing the organ with more blood at a time when it is needed. Histamine, or a like substance, is found in many tissues and is known to cause marked dilatation of capillaries and arterioles. It is believed that histamine or a like substance may account for the fall in blood pressure encountered in traumatic shock, as large amounts of it are found in mutilated tissue. On the other hand, the secretion of the suprarenal glands and of the posterior lobe of the pituitary gland causes vasoconstriction with a resulting rise in blood pressure.[5] Digitalis has a marked vasoconstricting action, while the vasodilating action of the nitrites causes a fall in blood pressure.

So far the regulation of circulation by changes in the caliber of vessels has been described as if the changes occurred in arteries only. However, there is an increasing amount of evidence [6] tending to show that capillaries and veins are also capable of dilatation and constriction under chemical or nervous stimuli. This conception constitutes a new perspective in the physiology of the circulation and may explain vascular phenomena hitherto not understood.[7] For example, in surgical shock the outstanding symptom is a serious fall in arterial pressure. This fall in arterial pressure is not due to a paralysis of vasomotor fibers, as was once believed, because in shock those fibers are in good condition. More recent experimental evidence indicates that the disturbance is in the area of the capillaries, those vessels being dilated, possibly by the action of histamine-like substances formed in damaged tissue.[8]

The vasomotor system of the nose, then, is a complex and unstable mechanism which involves a balance between arteriomotor, venomotor

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4. Bayliss, W.: Ergebn. d. Physiol. **5**:319, 1906.

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and capillomotor nerve fibers and chemical substances exerting regulating influences. It is a system that is as yet not entirely analyzed either anatomically or physiologically.

EFFECTS OF SALTS, DRUGS, VITAMINS AND HORMONES ON THE VASOMOTOR MECHANISM OF THE NOSE

The simplest and most direct proof of the presence and action of vasomotor nerves is obtained by direct inspection. Blanching of an organ—unless it is muscular, in which event mechanical factors must be considered—is interpreted as vasoconstriction. Similarly, reddening may be interpreted as vasodilatation. As the interior of the nose is not muscular, direct inspection was the method used to detect vasomotor

TABLE 1.—*Salts*

Compound	Dosage	Method of Administration	Vasomotor Effect	Duration of Effect
Calcium gluconate	20 grains (1.3 Gm.) q.i.d. for 6 weeks	Oral	None	
Calcium chloride	1 Gm.	Intravenous	Vasoconstriction	Transitory
Compound solution of iodine	8 minims (0.5 cc.) t.i.d. for 6 weeks	Oral	Vasodilatation	Cumulative effect, which ceased from 24 to 48 hours after ingestion
Sodium chloride	60 grains (3.90 Gm.) b.i.d. for 6 weeks	Oral	None	
Sodium chloride	500 cc. physiologic solution or 4.25 Gm. sodium chloride	Intravenous	None	
Pills of ferrous carbonate	5 grains (0.324 Gm.) q.i.d. for 6 weeks	Oral	None	
Amyl nitrite	.....	Inhalation	Vasodilatation	Transitory

action. Obviously this method does not lend itself to quantitative study, but this was not essential for this particular work. Blanching of the nasal mucosa and shrinking of the turbinates were interpreted as vasoconstriction, and reddening of the nasal mucous membrane with swelling of the turbinates was interpreted as vasodilatation, it being remembered that the turbinates contain erectile tissue.

For the purposes of clarity and conservation of space the accompanying tables are submitted to present the observations of the effects of inorganic compounds, organic compounds and hormones on the vasomotor mechanism of the nose. Each experiment was repeated on ten persons, some of whom had organic diseases, including nasal and vasomotor disturbances. The drugs chosen were those commonly used in therapeutics and are representative of the chemical classification to which they belong. The usual therapeutic dosages were employed.

From these observations it must be concluded that in normal persons but few drugs, salts, hormones or vitamins have effects on the vasomotor mechanism of the nose and that even these effects are transitory.

TABLE 2.—*Organic Compounds*

Compound	Dosage	Method of Administration	Vasomotor Effect	Duration of Effect
Quinine	5 grains (0.324 Gm.) t.i.d. for 6 weeks	Oral	None	
Atropine	1/150 grain (0.00043 Gm.) t.i.d. for 1 week	Oral	Vasoconstriction	Effect not evident 12 hours after last dose
Cocaine	2% solution	Topical application	Vasoconstriction	Transitory
Ephedrine	5% solution	Topical application	Vasoconstriction	Transitory
Histamine	.....	Topical application	Vasodilatation	Transitory
Digitalis	3 grains (0.195 Gm.) t.i.d. for 3 days	Oral	Vasoconstriction	Transitory
Solution of acetylsalicylic acid	10 grains (0.65 Gm.) t.i.d. for 1 week	Oral	None	
Sodium bicarbonate	30 grains (1.95 Gm.) t.i.d. for 1 week	Oral	None	
Dilute nitrohydrochloric acid	15 minims (0.92 cc.) t.i.d. for 6 weeks	Oral	None	

TABLE 3.—*Glandular Extracts*

Compound	Dosage	Method of Administration	Vasomotor Effect	Duration of Effect
Epinephrine	Solution 1 : 1,000	Topical application	Vasoconstriction	Transitory
Epinephrine	15 minims (0.92 cc.) solution 1 : 1,000	Subcutaneous	Vasoconstriction	Transitory
Posterior pituitary lobe	0.0005 Gm.	Subcutaneous	Vasoconstriction	Transitory
Thyroid extract	½ grain (0.032 Gm.) t.i.d. for 6 weeks	Oral	None	
Whole ovarian extract	5 grains (0.32 Gm.) t.i.d. for 6 weeks	Oral	None	
Corpus luteum	5 grains (0.32 Gm.) t.i.d. for 6 weeks	Oral	None	
Parathyroid	1/150 grain (0.00043 Gm.) t.i.d. for 6 weeks	Oral	None	
Insulin*	5 units per day	Subcutaneous	None	

* These observations were made on four patients who were diabetic.

TABLE 4.—*Vitamins*

Compound	Dosage	Method of Administration	Vasomotor Effect	Duration of Effect
Cod liver oil, vitamin A	1 tablespoonful b.i.d.	Oral	None	—
Viosterol, vitamin D	10 minims (0.6 cc.) t.i.d. 100 D, N. N. R.	Oral	None	—
Halliver oil, vitamin A	1 pellet (6,000 units) t.i.d.	Oral	None	—
Citrus fruits, vitamin C	250 cc. orange juice t.i.d.	Oral	None	—
Fresh vegetables, vitamins A, C, E	Diets rich in colored fresh vegetables	Oral	None	—
Yeast, vitamins B, E	1 cake t.i.d.	Oral	None	—

Many of the same preparations were administered to persons with vasomotor nasal disease, i. e., hyperesthetic rhinitis and hay fever, and the effect on the vascular mechanism of the nose was studied. The observations corresponded to those tabulated for normal persons; only transitory responses were noted, principally through topical applications of solutions of cocaine, epinephrine or ephedrine.

TABLE 5.—*Mechanical Agents*

Method of Stimulation	Vasomotor Effect	Duration of Effect
Mucous membrane of septum stroked with probe.....	Pale streak followed by hyperemic streak	—
Mucous membrane of turbinates stroked with probe....	Pale streak followed by hyperemic streak	—
Distant pain stimulation (pinching fingers, etc.).....	No effect	—

TABLE 6.—*Variations in Color of the Nasal Mucosa*

Clinical History	Number of Persons Examined	Color of Nasal Mucosa
Arteriosclerosis with hypertension, systolic pressure varying from 180 to 225	15	9 normal color 4 red 2 pale
Essential hypertension; systolic pressure from 180 to 210.....	10	7 normal color 1 red 2 pale
Coronary thrombosis with infarction of cardiac muscle.....	10	8 normal color 2 red
Buerger's disease (all patients had amputation of one lower extremity)	3	2 normal color 1 pale
Raynaud's disease	1	1 normal color
Vagotonia associated with dementia praecox.....	5	3 normal color 2 pale
Surgical shock	5	5 normal color
Traumatic shock	5	3 normal color 1 red 1 pale
Excitement, persons examined at horse races.....	10	8 normal color 1 red 1 pale

COLOR OF THE NASAL SEPTUM IN DEMONSTRATED VASCULAR DISEASE OR GENERAL VASOMOTOR DISTURBANCE

Table 6 was compiled for observations of the septal mucous membrane in persons with systemic vascular disease or general vasomotor disturbances, such as arteriosclerosis with hypertension, essential hypertension, coronary thrombosis, Buerger's disease, Raynaud's disease, vagotonia, surgical shock, traumatic shock and excitement.

The three types of color of the septal mucous membranes were found in the sixty-four persons studied in about the same proportion as observed in normal persons.[2]

CONCLUSIONS

1. The circulatory system of the nose is held in balance by vasomotor nerves and by chemicals. This balance is unstable. Little is known of its anatomy or physiology.

2. A few salts, drugs, hormones and vitamins have demonstrable effects on the vasomotor mechanism of the nose in normal persons and in patients with vasomotor nasal disease. The effects demonstrated were only transitory.

3. No relationship between the color of the nasal septal mucosa and demonstrable systemic vascular diseases could be demonstrated.

4. Until further light has been thrown on the anatomy and physiology of the vasomotor mechanism, claims of direct, specific therapy on this mechanism must be guarded. Beneficial results obtained by the ingestion of vitamins, etc., must be considered as general tonic effects rather than interpreted in terms of the vasomotor mechanism.

CHANGES IN THE LYSOZYME CONTENT OF THE NASAL MUCUS DURING COLDS

ANDERSON HILDING, M.D., PH.D.
DULUTH, MINN.

There is an enzyme normally present in the nasal mucus called lysozyme, which is a powerful antiseptic. The present article is a preliminary report of the changes in the lysozyme content of the nasal mucus during the course of the common cold. This study is one of a series on the physiology and pathology of the defenses of the nose begun some years ago in connection with investigations of the common head cold. Most of the studies on the mechanical defenses have already been published.

Lysozyme was first reported by Alexander Fleming [1] in 1922. It is found in most of the tissues and fluids of the body. It has been studied by a number of workers, most of whom are Europeans. Wolff [2] has worked on the chemistry; Hoder,[3] on its relation to immunology; Oluf Anderson,[4] on its importance in xerophthalmia and vitamin A deficiency; Goldworthy and Florey,[5] on the lysozyme action of intestinal mucus; Corper,[6] on the relation to tuberculosis; Ridley [7] and Cavka and Prica,[8] on the changes in lysozyme content of the tears in various

From St. Luke's Hospital.

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diseases of the eye. There have been a number of other studies reported on still other phases. Fleming[9] in 1929 gave a good summary of the facts known up to that time.

MATERIAL AND TECHNIC

The material consists of seven cases of infection of the upper respiratory tract. Samples of nasal secretion were collected from each patient daily or oftener. The relative power of each sample to destroy bacteria by lysis is given, shownig the changes that occur during a cold.

Two strains of bacteria, 9 and 12, were used for determining the power of lysis exhibited by the mucus. These strains were air-borne and belonged to the Sarcina group. No attempt was made to identify them more closely.

To make determinations, the samples of mucus were each diluted in a series of test tubes, as indicated in the tables, and a given amount of a heavy bacterial suspension was added to all the tubes. The suspension was sufficiently heavy so that after it had been added to the dilutions of mucus, the edge of a sheet of paper held against the test tubes could not be seen through the contained material. The tubes were incubated in the earlier determinations, but it was found that after a short time the heat caused the mucus to become so opalescent that readings became difficult and uncertain. This is the reason why many of the tubes containing the lowest dilutions, as reported in some of the tables, did not become entirely clear as would be expected. Therefore all the later determinations were made from tubes placed in the refrigerator. The time required for lysis was much longer with the refrigeration method, but the end-results were far superior because the solutions remained clear after lysis had taken place.

The clearing of the bacterial suspension began first, naturally, in the tube containing the highest concentration of secretion. Before it had advanced considerably in this tube the clearing had also begun in the tubes next in order, and before the first tube was entirely cleared the next several tubes were well advanced. It was of considerable interest to note that the tubes which were about half cleared showed definite clumping of the bacteria. In other words, the bacteria seemed to agglutinate to a certain extent before becoming dissolved.

The controls used were not the same in all experiments. For that reason the different tables are not all directly comparable, but each set of secretions examined constitutes a complete experiment in itself, and the relative findings in the different specimens are comparable. In the first experiments the degree of lysis was estimated with the eye, and the figures given are, therefore, only approximately correct. This method of estimation was used in testing the samples from cases 1 and 2 only. In later determinations a battery of eight control tubes, containing varying amounts of accurately measured quantities of bacterial suspension, was used for comparison. The figures from these later experiments are therefore much more nearly accurate.

One sample of normal nasal mucus was examined, together with each of the later determinations in order to have a normal standard of value.

The progress of symptoms was determined subjectively rather than objectively. The patient was closely questioned, and daily notes were made as to how he felt. This seemed to give a better index of progress than inspection of the nasal mucosa.

The results obtained are shown in tables 1 to 4.

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9. Fleming, Alexander: Arris and Gale Lecture on Lysozyme, a Bacteriolytic Ferment Found Normally in the Tissues and Secretions, Lancet **1**:217, 1929.

OBSERVATIONS

CASE 1.—The symptoms were as follows:

Feb. 18, 1933: At 10 p. m., on leaving a movie, the patient felt hot, sweaty and slightly stuffy. He remarked that he felt as though a cold were coming on.

February 19: At 4:30 a. m. the throat was slightly sore, and the right side of the nose felt stuffy. At 6:00 a. m. the first secretion from the right side appeared. The entire specimen was collected and put in the refrigerator (speci-

TABLE 1.—*Comparative Lytic Power of Specimens of Nasal Mucus Collected One Week Before the Onset of a Cold* *

Feb. 11, 1933	Dilutions	½ Hour	1⅙ Hours	2⅙ Hours	3½ Hours	6 Hours
Mucin a	1:10	50	100	100	100	100
	1:20	33	75	100	100	100
	1:40	..	25	75+	100	100
	1:80	..	..	75+	90	100
	1:175	..	..	50	50	66
	1:325	..	..	50	60	90
	1:650					
	1:1,310					
Tears	1:10	50	100	100	100	100
	1:20	33	75+	100	100	100
	1:40	..	50	100	90	100
	1:80	..	..	50	75	100
	1:175	..	30	60	75	100
	1:325	..	..	..	?	?
	1:650	..	..	..	?	33
	1:1,310					
Feb. 18, 1933		½ Hour	1 Hour	2⅙ Hours		5⅙ Hours
Mucin b	1:10	25	50	75		100
	1:20	25	33	66		100
	1:40	25	33	50		100
	1:80	..	25	33		90
	1:150	..	25	33		75
	1:300	..	..	?		50
	1:600	..	..	..		33
	1:1,200					
Tears	1:10	75	100	100		100
	1:20	50	100	100		100
	1:40	33	75	100		100
	1:80	25	50	75		75
	1:150	25	33	50		100
	1:300	..	25	33		50
	1:600	..	?	25		33
	1:1,200	..	..	..		25

* Results of observations on case 1. Strain 9 was used in bacterial suspension. Specimen *a* was obtained one week before specimen *b*. The figures at the left show the dilutions of the material tested in each of eight tubes. Those across the top indicate the time elapsed at each reading of the tubes after the dilution of mucus had been added to the bacterial suspension. The other figures indicate the percentage of clearing of the bacterial suspension. Tears were used as a control. The second specimen showed less lytic power than the first as compared with tears from the same patient. For instance, at the end of two and one-sixth hours, the first two dilutions of specimen *a* were entirely cleared whereas none from specimen *b* cleared in that time. The results of the tests made on specimens collected during the cold are given in table 2.

men *a*). The secretion was scanty all day. The cold was confined to the right side. Specimen *b* was collected at noon, *c*, at 4 p. m., and *d*, in the evening. The amount secreted was greater in the evening.

February 20: There was a moderate discharge all day, mostly from the right side. Specimen *e* was collected at 9 a. m. Specimen *f* was collected at 4 p. m. The patient felt badly. There was moderately profuse watery secretion.

February 21: The cold was slightly better; there was moderate discharge. The irritation seemed to be largely confined to one spot on the right. This side

TABLE 2.—*Comparative Lytic Power of Specimens of Nasal Mucus Collected During a Head Cold* *

Feb. 20, 1933	Dilutions	½ Hour	1 Hour	2 Hours	3 Hours	4 Hours
Specimen a	1:10	25	50	75	75	66
	1:20	?	25	50	50	50
	1:40	..	..	25	25	25
	1:80	..	..	..	..	?
	1:150					
	1:300					
Specimen b	1:10	25	33	33	66	75
	1:20	25	25	25	50	66
	1:40	..	..	?	25	50
	1:80					
	1:150					
	1:300					
Specimen c	1:10	33	50	90+	90+	90+
	1:20	25	25	75	90+	90+
	1:40	..	?	33	50	75
	1:80	..	..	?	30	33
	1:150	..	..	..	..	25
	1:300					
Specimen d	1:10	?	25	75	75+	90
	1:20	..	?	50	50	75
	1:40	..	..	33	25	2 broken
	1:80	..	..	?	?	25
	1:150	..	..	..	?	25
	1:300					
Specimen e	1:10	25	25	75	90	75
	1:20	?	?	50	75—	50
	1:40	..	..	33	50	33
	1:80	..	..	..	?	
	1:150					
	1:300					
Feb. 25, 1933		**½ Hour**	**1 Hour**	**2¼ Hours**	**3¼ Hours**	
Specimen f	1:10	?	25	50	75	
	1:20	?	?	33	50	
	1:40	..	?	?	25 ?	
	1:80					
	1:150	..	..	..	50	
	1:300					
Specimen g	1:10	75	100 ?	100	90	
	1:20	50	75	90	90	
	1:40	?	33	66	75	
	1:80	..	25	33	50	
	1:150	..	25	50	66	
	1:300	..	25 ?	50	50	
Specimen h	1:10	75	100	90	90	
	1:20	50	75†	75	90	
	1:40	?	25	33	75	
	1:80	..	33	25	50	
	1:150	..	50	75	75	
	1:300	..	33	33	50	
Specimen i	1:10	66	75	90	90	
	1:20	50	75	90	90	
	1:40	33	33	66	75	
	1:80	?	25	50	50	
	1:150	..	33	25	66	
	1:300	..	?	33	33	
Specimen j	1:10	100†	100†	75	75	
	1:20	100†	100†	75	75	
	1:40	50	75	75	75	
	1:80	25 ?	66	75	75	
	1:150	..	50	66	90	
	1:300	..	33	50	66	

* Results of observations on case 1. Strain 9 was used in the bacterial suspension. Specimens *a*, *b*, *c*, *d*, *e* and *f*, collected during the first two days of the attack, show a greatly reduced power of lysis. Specimen *g*, collected on the third day, was much more potent, as were all the specimens that followed. There was a slight improvement of symptoms on the day that specimen *g* was collected, and a marked improvement on the following day.

All of the tubes represented in these columns became clouded by the warming of the mucin in the incubator. The lysis was much more complete than the figures indicate. When similar dilutions of mucin were incubated for several hours without any bacterial suspension, they, too, became somewhat clouded. It was on this account that in later experiments the tubes were kept in the refrigerator instead of in the incubator.

† Tube contains considerable clouding from the mucin flakes. It seems to show no bacterial suspension between.

remained closed and the left remained open. At 1:00 p. m. specimen *g* was collected. The secretion was still profuse and watery.

February 22: The left side was involved also. The patient felt decidedly better. The left side never became bad. Specimen *h* was obtained.

February 23: The patient went back to work. The nose was practically dry; the cold was gone. Specimen *i* was obtained.

February 24: The patient felt fine. There was slight crusting of the nose. It was difficult to obtain specimen *j*. It was done by having the patient breathe cold air deeply and rapidly.

It happened by mere chance that a specimen of mucus had been tested against tears a week before, and a second specimen the day before the onset of the cold. The technic used in both instances was the same; hence the results should be comparable.

The second specimen, collected six hours before the onset of the first symptoms and eighteen hours before the first secretion, showed a distinct reduction in lytic power as compared with the one examined a week earlier, despite the fact that there was no more than the normal amount of secretion. The phenomenon was, therefore, not one of dilution by an increased quantity of secretion.

The cold lasted four days during which there was a profuse flow of watery secretion. Four specimens were collected on the first day, two on the second, one each on the third and fourth and one each on the next two days, subsequent to the cold. All specimens from the first two days exhibited a marked reduction in lytic power as compared with earlier and later specimens. The specimen taken on the third day had the greatest lytic power of any. At this time the patient thought that he felt slightly better, but he still had a profuse and watery discharge. He was sure that he felt better on the fourth day, and on the fifth day he returned to work. His discharge was scanty and thick on that day. On the sixth day there was so little secretion that it was difficult to obtain a specimen. The specimens collected on the fourth, fifth and sixth days all showed great lytic power, but not as great as that taken on the third day. Again this sudden increase of lytic power on the third day could not have been due to a corresponding decrease in volume of secretion because the discharge was about as great on the third as on the second day. Moreover, the specimens taken on the fifth and sixth days, when the secretion was scanty, showed somewhat less lytic power than the one taken on the third. Hence, the greatest potency was found in the specimen taken at the height of secretion and just preceding improvement of the symptoms.

CASE 2.—The symptoms were as follows:

March 3, 1933: The patient began to sneeze, and there was a moderate nasal secretion. A head cold had definitely begun. Specimen *a* was collected.

March 4: She was ill in bed all day with malaise and sneezing. There was scanty to moderate nasal secretion. Specimen *b* was collected.

March 5: There was much more nasal discharge, and the nasal obstruction was marked. Specimen c was obtained.

March 6: The cold was practically gone on awakening. There was almost no secretion. Specimen *d* was collected with difficulty.

The cold lasted three days. One specimen was collected on each of the three days (specimen *a, b* and *c*) and one (*d*) on the following day. The specimens from the first two days (*a* and *b*) showed a marked reduction as compared with specimen *d*. The secretion was moderate

TABLE 3.—*Comparative Lytic Power of Specimens of Nasal Secretion Collected on Successive Days of a Cold* *

	Dilutions	1 Hour	2 Hours	3 Hours+	4 Hours+	5 Hours
Specimen a	1:10	?	75	75	90	75
	1:20	?	50	50	75	75
	1:40	..	?	33	50	50
	1:80					
	1:160					
	1:320					
Specimen b	1:10	75	75	75+	75+	75+
	1:20	50	75	75	90	75
	1:40	?	..	50	75	66
	1:80	?	..	?	?	?
	1:160	..	..	..	?	?
	1:320					
Specimen c	1:10	75	75	100	90	75
	1:20	50	66	66	75	75
	1:40	50	50	50	66	66
	1:80	?	..	?	33	50
	1:160	..	..	..	?	50—
	1:320	..	..	..	?	33
Specimen d	1:10	100	100	100	100	100
	1:20	100	100	100	100	100
	1:40	50	66	75+	75	75
	1:80	33	50	50	50	50
	1:160	50	66	66	66	66
	1:320	33	50	33	50	50

* Results of observations on case 2. Strain 9 was used in the bacterial suspension. Specimens *a, b* and *c* show a marked reduction in lytic power in all dilutions as compared with specimen *d*. This can be seen readily from the results at the end of the first or second hour. Specimen *d* was collected the day that the symptoms largely subsided.

in amount on these days. Specimen *c*, taken on the third day, when there was more secretion, showed a slightly improved lytic power. The cold was gone on the fourth day, the secretion scanty and the lytic power high.

Case 3.—The cold in this instance lasted for six days. It followed exposure on a fishing trip. The first symptom was a "raw throat," but after the appearance of the rhinitis this symptom was not prominent.

Five specimens of secretion were collected, beginning with the second day. Each was tested in five different dilutions together with one specimen of normal secretion. Specimens *a* and *b*, collected on the second and third days, showed a definite reduction as compared with later specimens. The patient felt quite ill during these days with general malaise and rhinitis. Specimen *c*, taken on the fourth day, showed an

increasing lytic power. The patient felt better on that day. The specimens taken on the fifth and sixth days showed a still greater increase, and the patient felt still better, although on the sixth day there was a very considerable amount of secretion. Specimen *e*, taken on the last day of the cold, gave even higher values than the normal control.

Case 4.—The symptoms were as follows:

April 19, 1933: The patient awoke with a sore throat but felt better during the day. He did not seem to have a cold, although the throat was raw. Specimen *a* collected in the evening was scanty.

TABLE 4.—*Comparative Lytic Power of Five Specimens of Nasal Mucus Collected on Successive Days Throughout an Attack of Laryngitis* *

	Dilutions	½ Hour	1 Hour	1½ Hours	2 Hours
Specimen a	1:10	75	90	90	90
	1:40	50	75	75	75+
	1:60	25	50	75—	75
	1:640	..	25 ?	25	25
	1:2,560	..	25 ?	25	25
Specimen b	1:10	75	75	75	75
	1:40	75	90	90	90
	1:60	75	90	90	90
	1:640	50	50+	75	75
	1:2,560	..	25+	50	50
Specimen c	1:10	75+	90	90	90
	1:40	75	90	90	90
	7:60	50	75	75+	90
	1:640	25	50	50+	75—
	1:2,560	..	25	50	25
Specimen d	1:10	75	90	90	90
	1:40	75	90	90	90
	1:60	50	75	75+	75+
	1:640	25	50+	75—	75—
	1:2,560	..	50	25+	25
Specimen e	1:10	90	90	90	90
	1:40	75	75+	90	90
	1:60	50	75	90	90
	1:640	50	50+	75—	75—
	1:2,560	25	50	50	50

* Results of observations on case 4. Strain 12 was used in the bacterial suspension. All the reactions were approximately equal. In this case there were almost no nasal symptoms.

April 20: The throat was still raw. There was a slight nasal secretion from time to time during the day. Specimen *b* was collected. The secretion was still scanty.

April 21: There were moderate sore throat and moderate laryngitis. Heavy mucopus came from the throat. There was little if any nasal secretion or obstruction. The patient complained of slight malaise. In the evening there was nearly complete aphonia, with slight nasal secretion. Specimen *c* was obtained.

April 22: The laryngitis was somewhat better. Still there were almost no nasal symptoms. Headache appeared in the evening. Specimen *d* was obtained.

April 23: The patient was still almost aphonic. The nose remained practically normal. There was slight cough in the evening. Specimen *e* was obtained. The patient did not at any time have a definite so-called head cold.

This was an attack of pharyngitis and laryngitis and not the usual head cold. At no time was there any abnormally great secretion from

the nose. On the other hand, there was nearly complete aphonia for at least two days. The entire attack lasted five days. Specimens *a*, *b*, *c*, *d* and *e* were collected from the nose on successive days. When tested for lytic power, they were all found to give essentially similar reactions. There was no apparent reduction in lytic power of the nasal secretion during this attack of laryngitis and pharyngitis.

Case 5.—This patient suffered from a cold that had not entirely ceased after ten days when the last specimen was taken. Sore throat and headache were prominent. Nine specimens were collected, one each day from the second to the tenth, inclusive. They were *a*, *b*, *c*, *d*, *e*, *f*, *g*, *h* and *i*. There was not as marked a difference in the lytic power of these specimens as in some of the former cases. Specimens *a*, *b*, *c* and *d* were somewhat less active than the remainder. There was a suggestion of increased lysis in specimen *e*. It was definite in *f*. The patient began to feel better on the sixth day, when specimen *e* was collected, and felt fine, but had much secretion on the seventh day, when *f* was collected.

Case 6.—This was a brief attack that began with a sore throat and was over on the fourth day. The sore throat continued during the first three days. Four specimens were collected, one on each day. Specimens *a* and *b* showed a moderately decreased lytic power as compared with *c* and *d*. Improvement began on the day that *c* was collected. The amount of secretion was still profuse.

Case 7.—This patient suffered from a protracted attack that lasted for eight days. Sore throat was prominent throughout. All the specimens were rather strongly lytic and compared favorably with a normal control. There was some slight variation. The first two were the least active. The specimen taken on the sixth day was the most active. On that day the patient felt very ill; but on the day following she felt much better, and she improved from that time on.

The thought occurred to me that perhaps this change in power of lysis of the nasal secretion might be due to some change in the reaction. Lysozyme is said to be highly sensitive to changes in reaction. A p_H determination was made of all the specimens from cases 3 and 7. There was no definite change corresponding to the great change in lytic power. In fact, it was nearly uniform in all specimens. A number of p_H determinations were made on specimens collected from several other persons some of whom had colds or laryngitis and some of whom were free from colds. Some of these specimens were tested immediately, some after two, three, four, five and six days and some each after two and three weeks. All except those tested immediately showed a fairly uniform reaction ranging between 8.3 and 8.9. The majority reacted at 8.4 or 8.5.

SUMMARY AND COMMENT

The results of the tests for lysozyme in the nasal secretion from seven cases of head colds are given as a preliminary report. Conclusions should be drawn with caution because the cases are so few. The findings in some, however, indicate a sharp reduction in the natural antiseptic properties of the nasal secretion during and preceding the invasive stage. If, in the further studies now under way, this proves to be a characteristic of a large percentage of head colds, it will be a very suggestive piece of information about this common ailment.

Three of the seven cases studied (cases 1, 2 and 3) showed this marked loss in power to destroy bacteria by lysis. In one of these there was a definite reduction six hours previous to the first suggestion of a cold and eighteen hours before the first discharge of secretion. (This was, of course, a single observation and like all single observations must be corroborated before it can be accepted at face value.) Similarly, a definite improvement in symptoms was preceded by an increase in the lytic power of the secretion, twelve hours before. In the other cases (2 and 3) the increase in lysozyme occurred on the same day as the beginning of subjective improvement.

Two cases (5 and 6) showed only a small increase in lysozyme on the day of the first improvement. These and the remaining two cases (4 and 7), which showed no definite alterations in lytic power during the attacks, were all characterized by continued sore throat and in one instance by aphonia also. In the first three cases (in which the lytic power was greatly reduced) sore throat was not a prominent symptom.

In none of the patients suffering from sore throat was there any tonsillitis, only pharyngitis or laryngitis occurred. Three of them had had the tonsils cleanly removed.

This change in lysozyme content is definitely not a dilution phenomenon. The lysozyme was found to be low in at least one sample of scanty secretion preceding an attack, and it reached its high point several times in samples taken during an excessive flow of watery secretion on or preceding improvement of symptoms.

CHOLESTEATOMA ASSOCIATED WITH AN ISOLATED PERFORATION IN SHRAPNELL'S MEMBRANE

J. R. LINDSAY, M.D.
CHICAGO

The importance of a perforation in the upper periphery of the tympanic membrane as contrasted with a central perforation is demonstrated by the statistics of Scheibe [1] and Schlittler,[2] who found that 91 per cent of the cases of epitympanic perforation were complicated with cholesteatoma, and that 100 per cent of their cases of chronic suppuration with fatal complications were cholesteatoma.

The frequency of a perforation in Shrapnell's membrane attracted the attention of Bezold,[3] who found that this type comprised 6 per cent of his cases of chronic suppuration. He observed that perforation in this area was almost always associated with a collection of epidermis in the aditus and antrum.

The observation that a perforation in the pars flaccida never occurred during genuine or secondary acute suppurations of the middle ear indicated a different etiology from that of perforation or destruction of the pars tensa.

The main theories which have been advanced during the past half century or more as to the etiology of aural cholesteatoma, and in particular that associated with an isolated perforation in Shrapnell's membrane, may be outlined as follows: The occurrence of cholesteatoma in the spaces of the middle ear is noted in the writings of the early pathologists—Cruhveilhiers,[4] Rokitansky [5] and Virchow.[6] The similarity in structure of cholesteatoma in this locality to that found elsewhere in the skull and in connection with the pia led to the conclusion that the etiology was the same. The theory that aural cholesteatoma is a

From the Division of Otolaryngology, Department of Surgery, the University of Chicago.

1. Scheibe: Ztschr. f. Ohrenh. **75**:196, 1917.

2. Schlittler: Die Lebensgefährlichkeit der verschiedenen Formen der Mittelohreiterung, Berlin, Julius Springer, 1921.

3. Bezold, F., and Siebenmann, F.: Textbook of Otology, translated by J. Holinger, Chicago, E. H. Colegrove Co., 1908. Ztschr. f. Ohrenh. u. f. Krankh. d. Luftwege **20**:5, 1890.

4. Cruhveilhiers: Anatomie pathologique du corps humain, Paris, J. B. Baillière, 1829-1842, vol. 2.

5. Rokitansky: Lehrbuch der pathologischen Anatomie, ed. 3, Vienna, Wilhelm Braumüller, 1855, vol. 1, p. 220.

6. Virchow: Virchows Arch. f. path. Anat. **8**:371, 1855.

true or primary tumor is therefore the earlier explanation. The name "cholesteatoma" comes from Johannes Mueller.

Gruber (1862) and Troeltsch (1869) were the first of the otologists to recognize the almost constant association of aural cholesteatoma and chronic suppuration and to express the view that the cholesteatoma was the result of the suppurative process. The arrival of the epidermis in the middle ear had not yet been explained.

To explain the origin of the stratified squamous epithelium in the middle ear Wendt [7] (1873) advanced the theory that under the influence of chronic suppuration the epithelium of the middle ear underwent a metaplastic change to an epidermis-like layer, which, as a result of a desquamative inflammatory reaction, produced the mass known as cholesteatoma. The recognition that cholesteatoma never developed in connection with the large central perforations, however, provided strong evidence against this explanation.

The theory of immigration was advanced by Bezold [8] and Habermann.[8] According to this theory the arrival of the epidermis in the middle ear is explained as an extension from the epidermis of the external canal, in an attempt by nature to heal over the defects left by suppuration and necrosis of the mucosa. The ingrowth of epidermis may not always result in the formation of cholesteatoma, but if limited in extent may result in simple healing of the defect as the suppuration clears up. The marginal perforation, which is more likely to occur in the severe types of otitis media which also destroy the mucosa of the middle ear, provides easy access of the epidermis to the spaces of the middle ear. The accumulation of exfoliated products is enhanced by the desquamative reaction of the invading epidermis in the presence of suppuration.

The most recent theory, advanced by Manasse,[9] is that the epidermis of the canal or tympanic membrane, in the presence of suppuration, may take on the activity of new growth and enter the spaces of the middle ear, not as an extension on the surface, but as an invasion of the depths of the mucosa in the manner of a new growth. Thus the process would be classed as a tumor rather than as nature's attempt to heal a defect, and as a tumor of postfetal origin rather than one from inclusion of embryonic cell rests.

Definite clinical evidence in support of the theory of primary origin has been present only in rare cases. Of one hundred thousand cases in Basel, Munich and Zurich none gave definite evidence of primary origin

7. Wendt, quoted by W. Brock, in Denker, A. and Kahler, O.: Handbuch der Hals-, Nasen- Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 7, p. 229.

8. Habermann: Arch. f. Ohren, Nasen, u. Kehkopfh. **27**:42, 1889.

9. Manasse, P.: Handbuch der pathologischen Anatomie des menschlichen Ohres, Munich, J. F. Bergmann, 1917.

(Nager [10]). The microscopic examination of temporal bones has so far not revealed any case of cholesteatoma which was definitely primary. A few cases of primary cholesteatoma in the ear have been reported by both pathologists and clinicians—Kuhn, Koerner, Lucae and Erdheim and more recently by McKenzie. The theory that all aural cholesteatomas may be primary has recently been supported by McKenzie.[11]

The great frequency with which cholesteatoma occurs in the ear (over one third of all chronic suppurations, according to Scheibe), compared with the rarity of intracranial cholesteatoma or the rarity of inclusion tumors elsewhere in the body, is difficult to explain on the basis of the theory of primary origin. The suggestion was made by McKenzie that the peculiarities of development of the upper periphery of the tympanic membrane are such as to provide an area in which inclusion tumors are likely to occur. The opinion of the majority of otologists since the time of Bezold has been that true primary cholesteatoma in the ear is so rare as to be of little practical importance.

The application of the theory of immigration to the cholesteatoma found with an isolated perforation in Shrapnell's membrane has required special explanation. As perforation in this area does occur in acute destructive suppurations the production of the perforation itself has had to be explained. Bezold noted that perforation in Sharpnell's membrane was frequently associated with a sinking in or cupping of the same area in the opposite ear, and also that it was frequently associated with involvement of the eustachian tube, as, for example, in the presence of hypertrophied adenoid tissue. He therefore ascribed the chief etiologic rôle to tubal occlusion, in consequence of which the pars flaccida, being less resistant than the membrana propria, becomes retracted and ruptures, allowing for the entrance of infection and the ingrowth of epidermis from the margins. According to Wittmaack [12] the sinking in or cupping of the pars flaccida may be so marked as to cause retention of the desquamated surface layers of epithelium within the depression, which in turn causes the depression to enlarge inward, slowly producing an increasing mass of desquamated epidermis in the spaces of the middle ear. Thus a "dry" cholesteatoma may be formed before the beginning of suppuration. He ascribed the chief etiologic rôle to an already present hyperplastic otitis media, which alone may produce the retraction of Shrapnell's membrane without the necessity of tubal occlusion. Sections were shown by Wittmaack depicting marked hyperplastic otitis media and also a mass of cholesteatoma in the epitympanum, with the epidermal

10. Nager: Ann. Otol., Rhin. & Laryng. **34**:1249, 1925.

11. McKenzie: Proc. Roy. Soc. Med. **24**:6, 1931.

12. Wittmaack: in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12.

lining continuous with the epidermis of the canal and the tympanic membrane. The conclusion was drawn that the hyperplastic otitis produced a bridge of connective tissue separating the recessus from the tympanum and caused the retraction of Shrapnell's membrane and the production of cholesteatoma in the manner just described. Politzer [13] was of the opinion that the majority of cases of perforation of Shrapnell's membrane resulted from a generalized suppuration of the middle ear.

In the case of the necrosing otitis occurring in acute infectious diseases—which, according to Ulrich,[14] were responsible for 33 per cent of the cases of cholesteatoma observed—the theory of immigration has been confirmed by clinical observation. It has been possible to observe the different phases of development of the cholesteatoma, the onset of otitis media, the rupture and melting away of the drum and the extension of the epidermis within the spaces of the middle ear in a few months. Nager [10] cited cases in which an operation on the antrum for the relief of mastoiditis and empyema was followed by epithelialization of the cavity, requiring the radical operation within a few months of the onset of the original scarlatinal otitis.

I recently observed such a case requiring the radical operation. The onset of disease occurred two years before with bilateral scarlatinal otitis, followed in about three weeks by a bilateral simple mastoid operation. The necessity for the radical operation for cholesteatoma was recognized a few months after the simple operation, but was refused. At the subsequent operation the mastoid cavities were lined by epidermis, forming the so-called false cholesteatoma.

Polvogt [15] reported a case in which, five weeks after the onset of scarlatinal otitis, the epidermis could be seen extending beyond the annulus tympanicus into the middle ear. Investigations of Ulrich showed that at least a large percentage of the cases of cholesteatoma which give no clear history of causal involvement are to be attributed to tuberculous otitis.

In contrast to the cholesteatoma which has developed through a defect in the pars tensa or from total destruction of the drum an opportunity is seldom afforded to observe the development of an isolated perforation in Shrapnell's membrane and to confirm by clinical observation the mode of development of the cholesteatoma.

Perforations have been observed to occur in acute conditions, e. i., influenza, in the pars flaccida, with the discharge of a small amount of

13. Politzer: Textbook of Diseases of the Ear, ed. 6, Philadelphia, Lea & Febiger, 1926.

14. Ulrich, quoted by Nager, F. R.: Ann. Otol., Rhin. & Laryng. **34**:1249, 1925.

15. Polvogt, LeRoy M.: Pathogenesis of Cholesteatoma, Arch. Otol. **9**:597 (June) 1929.

pus from Prussak's space; but in chronic suppuration with perforation in this area there is usually no history of acute symptoms or of causal involvement to mark the onset.

Three cases of cholesteatoma with an isolated perforation in Shrapnell's membrane have been selected as representing different types of the clinical picture and, because of features presented, as having a bearing on the etiology.

REPORTS OF CASES

CASE 1.—R. A., a man, aged 40, complained of increasing deafness in the left ear. There was a history of intermittent nasal obstruction and frequent colds in the head and sore throat since childhood. The left ear had ached and discharged frequently since childhood. The hearing had become increasingly worse during the last few months. The right ear had also discharged frequently in former years.

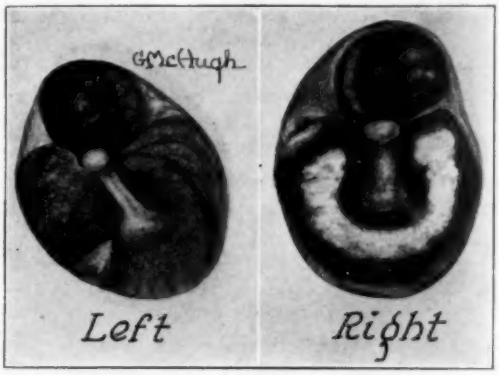

Fig. 1 (case 1).—Both ear drums after the left ear had healed. The pars tensa is intact on both sides; a crescent-shaped calcareous plaque is present in the right ear. The large perforation above the short process of the malleus opens into a small completely epithelialized cavity on both sides.

Examination revealed the tympanic membrane of the right ear moderately retracted, with a crescent-shaped calcareous plaque. The short process and the handle of the malleus were visible. At the site of Shrapnell's membrane was a large, deep, cuplike cavity lined with clean white epidermis, but without communication with the tympanic cavity. In the left ear the membrana propria was intact, but was moderately retracted and scarred. A mass of crusted material and discharge was present in Shrapnell's area, which, on removal, exposed a large perforation leading into the epitympanum. Fetid pus and cholesteatoma filled the opening.

A mass of cholesteatoma about the size of a large pea was removed through the perforation. Treatment by means of attic irrigations and removal of granulation tissue resulted in the ear becoming completely dry in about two months. It was evident from the size of the openings on both sides that there had been some erosion of the lateral bony margin of the epitympanic space. After the healing of the left ear was complete, the epidermis-lined cavity in the lateral epitympanic recess was little larger than the cavity in the same area in the opposite ear (fig. 1).

Comment.—It was evident that the extent of invasion of the recess by the epidermis was almost identical on both sides, but that retention of exfoliated products and persistence of suppuration had occurred only on the left. Whether the epidermis had extended inward after the perforation was already present or whether the cholesteatoma had developed as a dry type (Wittmaack) is a matter for speculation.

The long history of repeated infections, coupled with the bilateral changes, leaves no doubt that the cholesteatoma developed secondary to the inflammatory changes. The not infrequent occurrence of an isolated perforation in Shrapnell's membrane and of epidermis in the recessus epitympanicus, with a history of long or repeated suppuration, as a bilateral finding is strong evidence of the secondary nature of the cholesteatoma.

CASE 2.—A. M. R., a girl, aged 6 years, was brought to the clinic for a routine examination of the nose and throat. There were no complaints and no history of disease of the ear, nose or throat, except one cold in the head a year. Examination showed moderate hypertrophy of the tonsils, adenoids (not causing obstruction) and a normal nose. Both tympanic membranes were intact. No treatment was advised.

About two and a half years later the child was again brought to the clinic on account of a slight discharge from the right ear for a few weeks. The history was as follows: The discharge was first noticed a few weeks before examination, but was slight and was disregarded for a time. On questioning the mother recalled that the child had complained of soreness in the ear on one occasion about five months previously, which had disappeared when a hot water bottle was applied. The mother was sure that there had never been a discharge from the ear in infancy or in later childhood and that there had been no complaint until five months previously. The child's hearing had always been good and her progress in school rapid.

Examination revealed the tympanic membrane of the left ear to be normal. The external canal of the right ear contained a scanty purulent discharge with a foul odor. The pars tensa was intact and not retracted, but lacked luster. The short process and handle of the malleus were visible. In Shrapnell's membrane was a small perforation, with a tiny polypoid granulation projecting. The nose appeared to be normal and the tonsils and adenoids appeared as on the previous examination. A physical examination gave negative results.

The ear showed no improvement after a period of conservative treatment except that the odor disappeared. Removal of the projecting granulation was followed repeatedly by its reappearance. The perforation was too small to permit irrigation through an attic cannula. A roentgenogram showed a normally aerated mastoid process on the left, and on the right a clouding of all the mastoid cells, with apparent destruction of the cells in the region of the antrum.

The mastoid process was opened through a curved incision 1 cm. behind the postauricular furrow. A few moderately large cells were found in the region of the tip. In the body of the mastoid the bone was diploic, and in the region of the antrum a pearly white soft mass of cholestatoma was uncovered. The mass measured about 1 cm. in the horizontal diameter and 1.5 cm. in the vertical, and

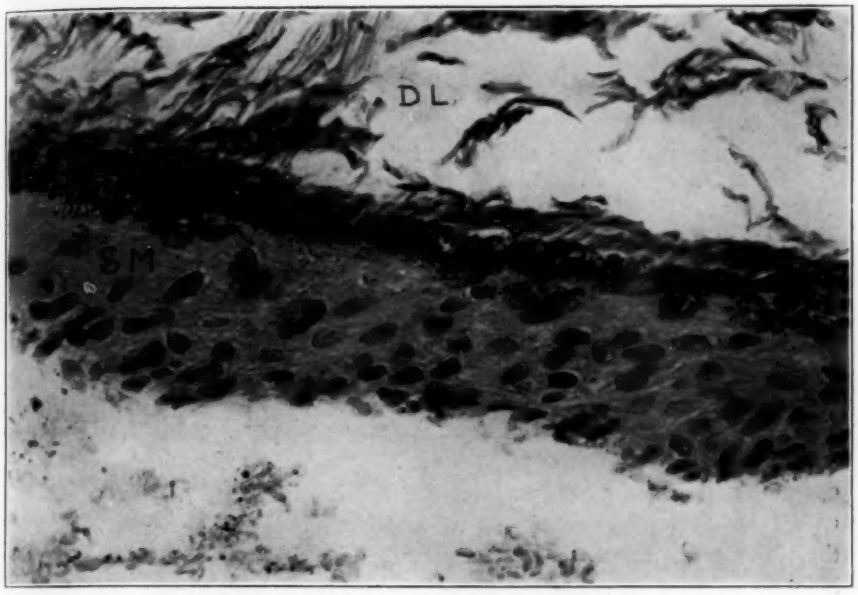

Fig. 2 (case 2).—The epidermal layer of the cholesteatoma; *SM* is the stratum malpighii, *SG* the stratum granulosum and *DL* some of the desquamated cell layers.

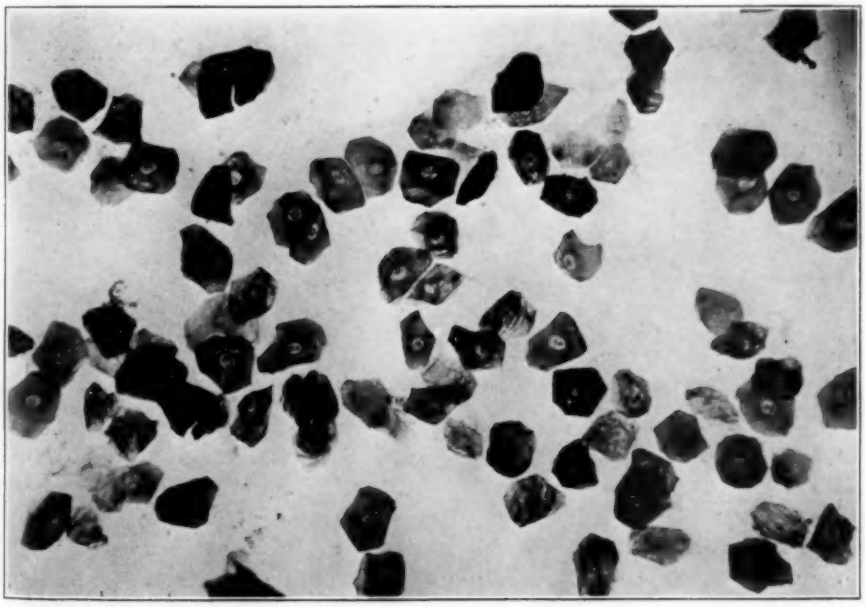

Fig. 3 (case 2).—The nucleated squamous cells forming in the concentric layers of the cholesteatomatous mass.

extended forward into the epitympanum. The modified radical operation was done, leaving the ossicles in place and not disturbing the matrix on the medial wall of the cavity.

The postoperative course and healing of the cavity were uneventful.

Comment.—The features in this case which have a bearing on the etiology may be reviewed as follows: Factors which predispose to involvements of the eustachian tube were absent. The occurrence of previous suppuration in the ear could be safely ruled out. The tympanic membranes were observed on two examinations less than three years previously, and were found normal. The left ear showed no abnormality at any time.

It may be safely assumed that the size and position of the cholesteatomatous mass in this case preclude the possibility of its having developed after the discharge first made its appearance. Also, the possibility of this mass having developed as a dry cholesteatoma subsequent to the previous examinations is scarcely credible, since such a process must be very slow in development and since factors which might indicate a change in the tubes or middle ears were absent until a few months before operation.

The suppurative process was apparently limited to the epitympanum, and the perforation was very small and apparently central. No evidence of active infection was present either within the cholesteatomatous mass or in the subepithelial tissues in the region of the antrum. Figure 2 shows the epidermal wall and figure 3 the flat nucleated cells from the cholesteatomatous mass.

The possibility of this cholesteatoma having developed by immigration from the canal seems remote, whereas there is definite evidence to indicate that the mass was primary in origin.

CASE 3.—A man, aged 20 years, had had a discharge from the left ear since the age of 2 years, except for a period of two years beginning at the age of 8. He had had frequent colds in winter and much stopping up of the nose in summer since childhood. Examination showed the right tympanic membrane to be normal. The left auricular canal contained pus. In the pars tensa was a small central perforation, posterior to the umbo, and in Shrapnell's membrane a small perforation with a tiny red granulation projecting.

Conservative treatment did not improve the condition, and a modified radical operation was done. A cavity lined with cholesteatoma occupied the regions of the antrum and aditus. Figure 4 shows part of the cholesteatoma matrix attached to the bony wall of the antrum.

Comment.—This case may be more typical of perforation of Shrapnell's membrane in that there is little definite evidence as to etiology. The early onset of the suppuration and the unilateral character suggest the explanation on the basis of inflammatory changes in the middle ear

Fig. 4 (case 3).—Matrix of cholesteatoma attached to the bone in the region of the antrum. *A* is the epidermal layer, *B* the subepithlial layer, *C* the cystic cavity lined by cuboidal cells, probably formed by the walling off of a part of the mucous membrane.

Fig. 5.—A section of a cholesteatoma removed from the mastoid process showing: *A*, the typical concentric arrangement of exfoliated cell layers; *B*, the epidermal layer, and *C*, connective tissue layer, with vessels and infiltration by round cells and leukocytes.

in infancy, with the resulting chronic suppuration and with cholesteatoma as a secondary development. However, the possibility of a primary origin of the cholesteatoma cannot be ruled out.

SUMMARY

The three cases presented of isolated perforation in Shrapnell's membrane illustrate different clinical types of the condition.

The first case illustrates the group with a history of long-continued tubal involvement and repeated suppuration of the middle ear, in which the production of attic cholesteatoma has taken place secondary to the inflammatory changes and in which the perforation in Shrapnell's area is apparently the residue of a previous generalized suppuration of the middle ear. The frequency with which bilateral involvement is present in these cases confirms the view that the cholesteatoma is secondary.

The second case is unusual in that there is definite evidence of the primary origin of the cholesteatoma. As there is seldom opportunity to observe such cases either before or at the time the disease first appears, the question arises as to whether primary cholesteatoma in the ear may be as rare as is supposed.

Case 3 illustrates the common clinical type in which there is little definite evidence as to etiology.

EXTERNAL FRONTO-ETHMOSPHENOID OPERATION

NEW MUCOSAL FLAP FOR CONTROLLING FRONTONASAL DRAINAGE AND GRANULATION TISSUE; REVIEW OF SPHENOID TECHNIC

EDWARD CECIL SEWALL, M.D.
SAN FRANCISCO

The use of the fronto-ethmosphenoid operation has been limited by me to cases of severity sufficient to balance its known imperfections. All the ethmoid lateral extensions can be obliterated by this technic. However, one must recognize that the frontal sinus is included in the operation. The responsibility for the proper attention to that cavity is at once assumed by the operator. Adequate drainage is essential. This has been the weak point in the method because, owing to the lack of bony support, the soft parts tend to be drawn toward the medial side and to block the frontonasal opening. If the frontal sinus heals before this blocking takes place or if a sufficiently large opening remains, all is well. The retention of disease in the frontal sinus without adequate drainage into the nose must lead ultimately to the formation of an abscess. This imperfection has long been recognized. It brought opprobrium on the classic endeavors of Killian even more than the disfiguration from his thorough and excellent technic.

Efforts made to obviate this difficulty have included the use of flaps, grafts and dilation and the long wearing of rubber tubes. None of these methods has proved satisfactory. One has to face the possibility of future and more radical surgical intervention in a small group of cases, in my experience about 6 per cent (two of the patients were tuberculous). While this percentage appears small, the fault remains and interferes with the free use of an operation that has much to recommend it, at least until a distaste for operative procedures is justified by other methods for relief.

It must be recognized that in the surgical handling of sinusal disease the rhinologist is asked to perform a task demanded of no other surgeon. Elsewhere in the body the suppurating cavity is allowed to close. The last frontier was the chest, and now costal resection has completed the picture—not so with the nasal sinuses. Cosmetic and technical considerations demand that in treatment their outward conformation be retained.

Demonstrated on a cadaver and at operation before the Research Study Club, Los Angeles, Jan. 15, 1934.

To keep the frontonasal duct patulous appears to be a difficult problem, and I now suggest a flap which seems to offer help to that end. After fashioning it many times on cadavers I was discouraged because of the friability of the embalmed tissue. Nevertheless, I had the good fortune to find it much easier of accomplishment on the living tissue. It appears to offer possibilities.

I have constructed another flap from the mucosa covering the medial wall of the ethmoid mass and its turbinal extensions. This flap, turned up at the roof of the exenterated ethmoid mass, appears to epithelialize and shorten the course of healing and prevent the formation of obstructing granulation tissue. The flaps are constructed and disposed as follows:

1. In my fronto-ethmosphenoid technic as heretofore described,[1] the mucous membrane of the agger region in the nose is exposed by window resection of the frontal process of the maxilla. In the new technic after

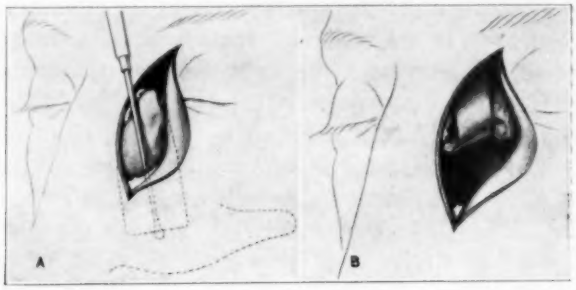

Fig. 1.—*A*, the nasal mucosa has been bared. The separation of the flap and the approximate extent of flap are shown. *B*, the flap of nasal mucosa attached above is turned upward to line the frontonasal opening and the sinus.

the frontal sinus, ethmoids and sphenoid have been attended to, the nasal mucosa is separated from the lateral wall of the nose. This is easily accomplished by passing a spatula down inside the exposed bony edge as shown in figure 1*A*. The spatula passes down on the nasal wall of the maxillary bone, and the mucous membrane is separated down to, or even onto, the inferior turbinal. The raised mucosa can be determined by looking into the nose through a speculum, and the position of the underlying spatula is seen as it is moved from above; a long sharp knife is introduced into the nose, and a horizontal cut is made through the mucosa down to the underlying spatula. This fashions the lower end of the flap. The anterior and posterior cuts are then made as shown in figure 2 *B*. The upper parts of the anterior and posterior incisions may be completed through the external wound. In working through

1. Sewall, E. C.: External Operation on the Ethmosphenoid-Frontal Group of Sinuses under Local Anesthesia, Arch. Otolaryng. **4**:377 (Nov.) 1926.

the nose or through the external wound the flap is raised and may be pulled upward. Still attached at its upper end, it is placed in the region of the frontal duct and plastered over as much of the frontal sinus as it will cover. A flap from 4 to 7 cm. in length, 2 to 3 cm. wide at the bottom and 1 to 2 cm. wide at the top may be gained. Free bleeding from the flap, an evidence of good blood supply in spite of ligation of the ethmoid arteries, has been noted.

2. Besides this flap considerable mucous membrane can be gained from the covering of the median wall of the ethmoid mass including the ethmoturbinals. Whatever can be secured may be thrown laterally and upward to cover the denuded under surface of the frontal bone, adjacent and lateral to the cribriform plate (fig. 2*A*).

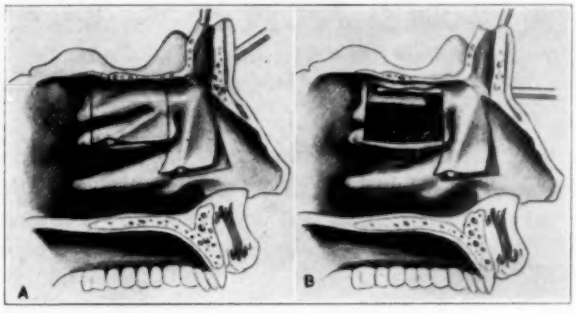

Fig. 2.—*A*, the preparation of the two flaps is shown from the nasal aspect. *B*, the ethmoid flap is being smoothed over the denuded surface of the ethmoid roof. The frontal flap is still represented in the nose.

OBLITERATION OF SINUS

When the condition of the frontal sinus is not satisfactory following a cosmetically perfect fronto-ethmosphenoid operation, fortunately a method is applicable that will take care of even desperate cases. I refer to obliteration of the sinus. This consists in the removal of the anterior and inferior walls of the frontal sinus, as outlined by Killian and others, and in the complete removal of even the orbital ridge which he so carefully retained for cosmetic reasons. It is surprising how little deformity remains after primary suture and healing when the sinus is not too large. The size must be measured by the true anteroposterior film. The work is usually accomplished through the fronto-ethmosphenoid incision which allows great freedom of access because of the movement of the soft parts.

When even this method was recognized to be inadequate because of the tuberculous process I packed the cavity with gauze and allowed it to fill by granulation after the removal of the anterior and orbital walls. Healing with comparatively slight deformity resulted.

REVIEW OF SPHENOID TECHNIC

In 1926 my article on the fronto-ethmosphenoid technic pointed out the necessity for the ligation of the sphenopalatine artery and the removal of the floor of both sphenoid sinuses when real drainage of those cavities is essential. I have always believed that such a radical technic is demanded only when the mucosa is so diseased that its removal is imperative. The ligation of the sphenopalatine artery was described. Time has justified the application of this technic, but has also suggested the helpful solution of a difficult problem. I never felt satisfied when ligating both sphenopalatine arteries, and I soon found that this was not necessary because both sphenoids are operated on from the one side and the procedure is satisfactory if the drainage is properly cared for. I have seen granulating, purulently diseased sphenoids defy partial removal of the floor and heal smooth and clean when the often difficult task of removing the floor was properly accomplished.

The relation of the vidian nerve to the floor of the sphenoid was brought out forcibly in a recent meeting of the Los Angeles Research Study Group. Dissecting under the direction of Dr. Barnhill, the class identified one vidian nerve lying not only free in the sphenoid sinus, but actually in the middle of its floor. Removal of the floor entailed its destruction. While this anomaly is unusual, it must be given serious consideration.

Stanford University Hospital.

Clinical Notes

HEMANGIO-ENDOTHELIOMA OF THE TEMPORAL BONE

JOSEPH A. SULLIVAN, M.B., TORONTO, CANADA

Careful search of the American, English and German literature yielded surprisingly little material and few completely reported cases of hemangio-endothelioma of the temporal bone. Frederick L. Jack, of Boston, reported a case in an adult.[1]

REPORT OF CASE

History.—A girl, 5 years of age, was admitted to St. Michael's Hospital on Dec. 8, 1932, complaining of severe pain in the left ear. According to the mother the child had been in perfect health until about the middle of October when she complained of pain in the left ear, which persisted and became progressively more severe. On the third day after the onset of the pain the mother noticed a gray, cauliflower-like growth deep in the external auditory canal. A physician removed a piece of the growth for microscopic examination. Subsequently he stated that he had received a report that it resembled cancer. Following the removal of a specimen for biopsy the tumor grew rapidly until it completely filled the auditory canal, and a thin, yellow, watery, foul-smelling discharge appeared. About this time the mother noted that the child's face was drawn to the right side and that she was unable to close the left eye. The left side of the face anterior to the ear became diffusely swollen and very tender. The child had increasing difficulty in swallowing solid foods and lost a great deal of weight.

On December 5 the patient began to vomit twice a day, and a firm swelling was noted along the inferior portion of the left mandible On December 6 a croupy cough developed, with abundant white frothy sputum.

When the patient was admitted to the hospital on December 8, the temperature was 97 F., the respiratory rate 20 and the pulse rate 84. The lungs, heart and abdomen were apparently normal.

Roentgen examination of both mastoids, with special plates to show the petrous apexes, revealed that the cells of the left mastoid were of pneumatic type and of average extent. There were no cells present of zygomatic distribution. The septums of the cells could be distinguished, but were slightly blurred. There was also a suggestion of decalcification involving the anterior wall of the sinus. The petrous apex on the right side was clearly defined, the petrosphenoidal suture standing out sharply. The foramen ovale and the foramen spinosum were clearly visible. Their margins were sharp. On the left side there was marked loss of definition in the region of the petrous tip, the foramina presenting blurred margins. The petrosphenoidal suture was practically obliterated. The obliteration of the bony landmarks of the petrous apex was highly suggestive of a destructive process, probably malignant (figs. 1 and 2).

From the Department of Oto-Laryngology, St. Michael's Hospital, and the Department of Anatomy, University of Toronto.

1. Jack, F. L.: Tr. Am. Otol. Soc. **12**:539, 1912.

On December 12 a radical mastoid operation was performed. The mastoid process was found to be filled with bleeding granular tissue which extended into all the cellular areas with complete destruction of the sinus plate, the tegmen over the middle and posterior fossas and the tegmen tympani. The middle ear and its contents were embedded in this mass of tissue, and the floor and internal and anterior walls were eroded. The posterior wall of the canal appeared as a sequestrum with the facial nerve in situ. A portion of the growth and of the bony sequestrum was removed for microscopic examination. Histologic examination

Fig. 1.—A lateral view of the left mastoid process showing the character and extent of the lesion.

revealed the following: The sections showed a variable structure. In places the tissue was composed of proliferating fibroblasts between which were numerous fine blood vessels, many lymphocytes and a small number of polymorphonuclear leukocytes (fig. 3). Elsewhere the tissue contained numerous loose clusters of spherical, deeply staining cells surrounded by zones of hemorrhage (fig. 4). These cells were also to be seen as masses within dilated blood channels (fig. 5), and in certain sections they appeared to spring from the endothelial lining of the blood channels and project into the lumen of the vessels as papillary projections with a fibrous tissue core (fig. 6). The stroma of the tumor was composed of moderately dense fibrous tissue arranged in a somewhat whorled fashion. The appearances were those

of a malignant neoplasm. The extreme vascularity of the growth, and the fact that the tumor cells occupied the blood channels and in places appeared to originate in the lining cells of such channels suggested that it was of endotheliomatous nature, and was to be classed as a malignant hemangio-endothelioma.

The patient was allowed to leave the hospital on Jan. 5, 1933. Her condition was fairly satisfactory; the facial paralysis persisted, but the pain had disappeared. When she was readmitted on January 22 for roentgen treatment, the paralysis of the left side of the face and soft palate was more evident. The upper region

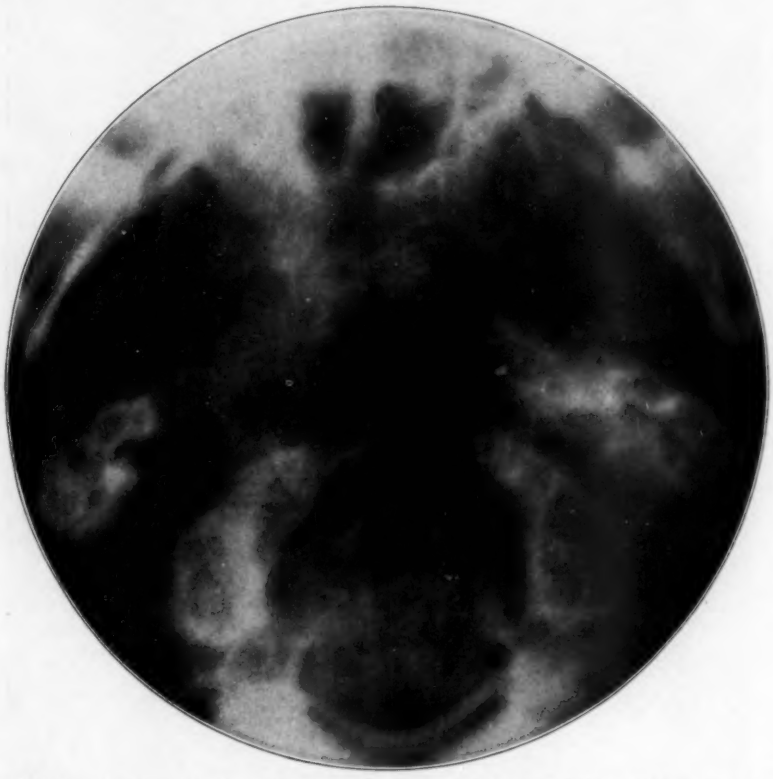

Fig. 2.—A view of the left mastoid process showing obliteration of the bony landmarks of the petrous apex.

of the face was not involved. The patient slowly became weaker and died on February 16.

Autopsy.—The brain weighed 1,150 Gm. There was no evidence of inflammatory change over the upper part of the brain or in the meninges. On removing the brain an irregular soft white tumor mass was observed involving the whole of the petrous portion of the left temporal bone. This mass was extradural for the most part, but had apparently infiltrated along the course of the seventh and eighth cranial nerves from the internal auditory meatus and had reached the surface of the left cerebellar hemisphere and pons. The tumor involving the petrous portion of the left temporal bone had eroded the bone completely, and appeared

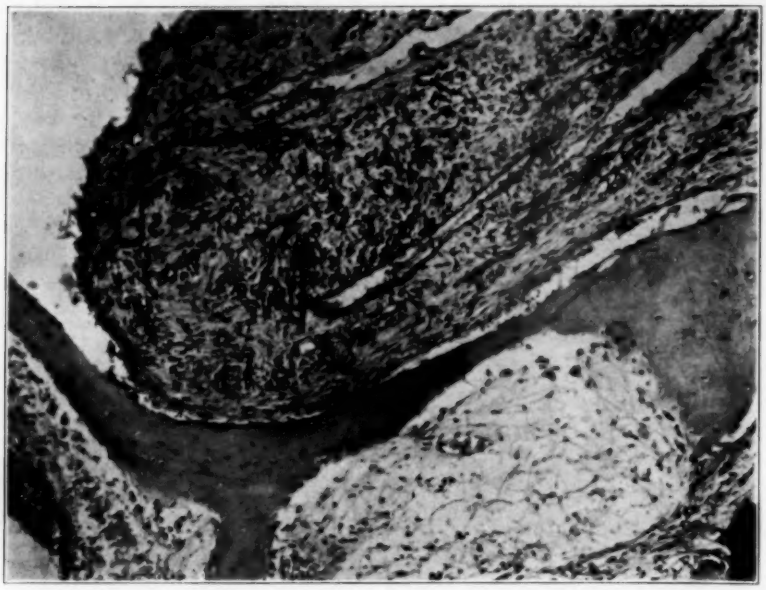

Fig. 3.—Proliferating fibroblasts, with numerous fine blood vessels, lymphocytes and some polymorphonuclear leukocytes.

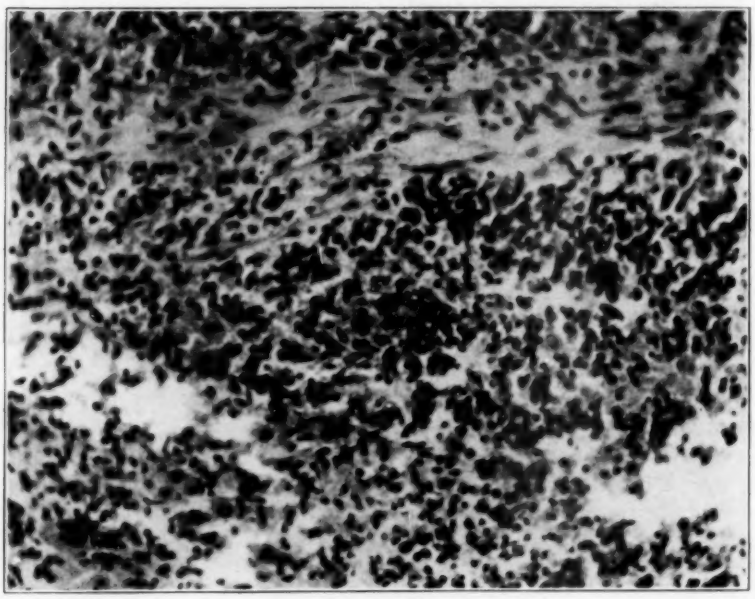

Fig. 4.—Loose clusters of spherical, deeply staining cells surrounded by zones of hemorrhage.

Fig. 5.—The spherical cells as masses in dilated blood channels.

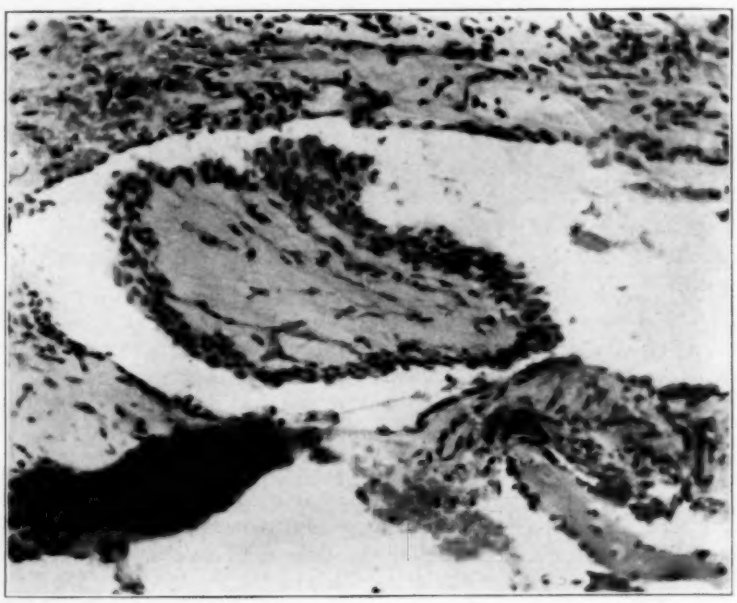

Fig. 6.—The spherical cells originating in the lining cells of the blood channels.

rubbery, with soft nodules of tissue extending into the greater wing of the sphenoid anteriorly and into the left border of the foramen magnum posteriorly. Medially it had encroached on the porous bone of the cavernous sinus. The petrous portion of the temporal bone and the tumor were removed intact. The tumor was found to have infiltrated downward through the cranial bones into the tissues of the neck and nasopharynx. This portion of the growth was soft and apparently necrotic. The left mastoid process had been completely excavated at operation.

Microscopic examination of the tumor revealed a fine network of connective tissue supporting masses of round and spindle cells with deeply staining nuclei. In some places the masses were compact; elsewhere the tumor cells were separated from each other as the result of edema. There were many well formed blood

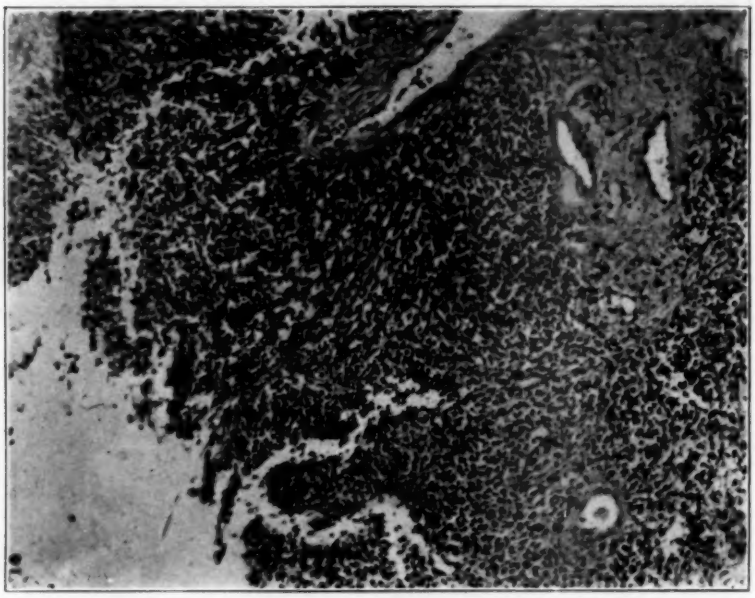

Fig. 7.—Specimens of the tumor obtained at autopsy showing masses of round and spindle cells, with no evidence of the formation of blood channels by the tumor cells.

vessels, a few of which showed thrombosis. There was no evidence of the formation of blood channels by the tumor cells (fig. 7).

COMMENT

The diagnosis of hemangio-endothelioma was based on the character of the tissue removed from the growth at operation. In the specimen obtained at autopsy the structure was definitely sarcomatous, and there was nothing to indicate an origin of the tumor cells from vascular endothelium. This change in type may be explained by the increased rapidity of growth with the resulting loss of cell differentiation. The pathologic diagnosis was: hemangio-endothelioma of the left petrous portion of the temporal bone; extension of the tumor into the great wing of the sphenoid and middle ear; extension of the tumor into the soft tissues of the neck;

a scar of a radical mastoid operation on the left side, and extension of the tumor to the surface of the left cerebellar hemisphere.

Treatment in such a case depends entirely on how early the diagnosis can be made. A few reports advocate complete removal of the temporal bone, while others suggest high voltage roentgen therapy. In a case such as I have presented, the futility of both of these procedures is evident. I believe that under the majority of conditions the treatment is only palliative.

Dr. William Magner, pathologist of St. Michael's Hospital, furnished the microscopic sections and Dr. W. Shannon provided the roentgenograms and interpreted them.

LOCAL ANESTHESIA IN RADICAL OPERATIONS ON THE MAXILLARY SINUS

A Simple Method of One Injection

BENJAMIN H. SHUSTER, M.D., AND W. CLYDE DECKER, D.D.S., PHILADELPHIA

It is frequently desirable to employ local anesthesia in a radical operation on the maxillary sinus. Ether and gas anesthesia interfere with the work of the surgeon, aside from having other contraindications. Rectal anesthesia has its objections and objectors. Even avertin by rectum does not often give complete anesthesia and has the objectionable feature of requiring elaborate preparations, such as an enema, weighing of the patient, calculation of the dosage, and a request for the proper solution, and it results in prolonged sleep of several hours following the operation. It is not without fatal outcome.

Certain methods of local anesthesia have been used with satisfaction. Infiltration has been employed with poor results; block anesthesia has given excellent results. The latter method requires injection at several points and since at times it is difficult to locate the various foramina for injection, the method becomes difficult.

The method we describe is one of a single injection into the infra-orbital foramen directly through the cheek. This was suggested by one of us (W. C. D.) who employs it as a routine in extracting teeth and in executing other work on the upper jaw, such as filling and grinding teeth, extirpation of pulp and curetting. He makes the injection into the infra-orbital foramen under the upper lip. He suggested that this method be tried in operations on the maxillary sinus.

One of us (B. H. S.) tried it successfully in several cases and found it much easier to locate the infra-orbital foramen directly through the cheek than under the lip. Anesthesia was excellent.

METHOD

It is helpful but not necessary to give the patient 3 grains (0.19 Gm.) of sodium amytal or pentobarbital sodium by mouth three hours before the operation and to administer a hypodermic solution of morphine, ¼ grain (16 mg.), and scopolamine, 1/150 grain (0.4 mg.), one-half hour before operation. When the patient is brought to the operating room the nose is packed with a 10 per cent

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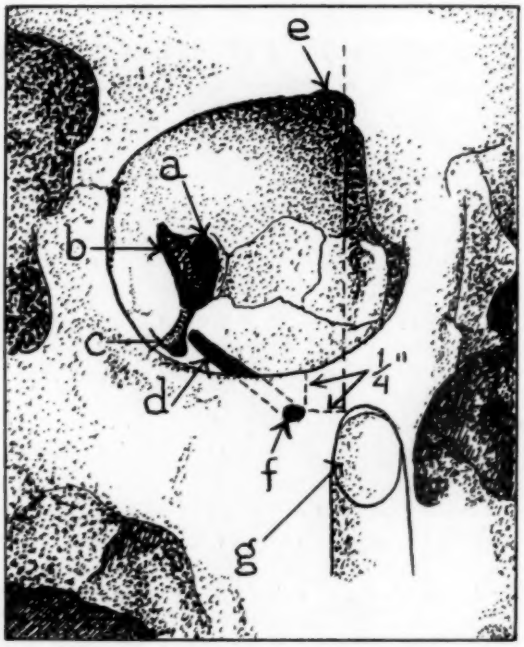
From the Oto-Rhinologic Service of Dr. George M. Coates, Graduate School of Medicine, University of Pennsylvania.

solution of cocaine with some epinephrine for nasal anesthesia and hemostasis. These packs are left in until one is ready to do the intranasal work. The injection of procaine hydrochloride, 2 per cent, is now made into the infra-orbital foramen. From 2.5 to 3 cc. of the solution with epinephrine is used. It is important that the solution be injected slowly, drop by drop, taking about three minutes to inject the entire quantity. The operation may be started as soon as the injection is completed.

The foramen can easily be found by palpation or by palpating the supra-orbital foramen or notch; the infra-orbital foramen is found about a quarter of an inch (0.6 cm.) outside the vertical line dropped from the supra-orbital notch and



Area of injection showing: *a*, optic foramen; *b*, supra-orbital fissure; *c*, infra-orbital fissure; *d*, area of distribution of injected fluid; *e*, supra-orbital notch; *f*, infra-orbital foramen; *g*, line of palpation, a fingerbreadth from the margin of the ascending process of the superior maxilla; the finger-tip, when rotated outward, touches the infra-orbital foramen.

about a quarter of an inch below the inferior orbital margin. These measurements were established by examining a number of skulls.

The forefinger is a good guide to finding the foramen. When the margin of the ascending process of the superior maxilla is felt the finger is rotated outward. The outer margin of the finger is in line with the foramen. By using the finger externally as a guide the needle is introduced under the lip and inserted into the foramen.

Another useful guide is the center of the patient's pupil. When the patient looks directly at the surgeon in front of him the foramen is located in the vertical line dropped from the pupil. These several guides for locating the foramen are

taken into consideration, because it is well to locate the foramen as directly as possible since with aimless search the needle scratches the periosteum as well as teases the inferior orbital nerve.

It is important to use a flexible needle which will not break (platinum or composition) and to inject the fluid slowly. The needle need not be inserted deeper than from ¼ to ½ inch (0.6 to 1.2 cm.) into the canal and should be directed slightly outward. One should always feel considerable resistance to the fluid when the injection is made. A rigid needle may penetrate into the orbit (should this occur the resistance to the fluid is immediately lost). We saw one such case, in which the lower eyelid swelled as a result. The swelling soon disappeared and there were no outward effects.

It seemed that the quantity injected could not all remain within the canal, but must be forced beyond the orbit. The good anesthesia produced also suggested that the solution had penetrated to the fluid of the main trunk of the second division of the fifth nerve and probably into the sphenopalatine ganglion. Through the courtesy of Dr. O. V. Batson, of the Graduate School of Medicine, University of Pennsylvania, dissecting material was available. A solution of methylthionine chloride, U. S. P. (methylene blue), was injected into the canal by the technic just outlined. The periorbita was then dissected to the posterior part of the orbit. The blue fluid was seen in the entire canal and its tributaries as well as in the region of the sphenopalatine fossa where it communicates with the zygomatic fossa at the inferior orbital fissure. In this region are located the second division of the fifth nerve and the sphenopalatine ganglion. In a second experiment the periorbita was first dissected and then the fluid was injected. The colored fluid could be seen slowly to invade the areas previously mentioned, and each drop injected could be seen forcing its way deeper into the canal. The regions of the superior orbital fissure and the optic foramen were entirely free from any colored fluid. Through these openings fluid would most likely enter the cranium. The solution apparently does not travel in that direction.

While we have only a few cases to testify to the effectiveness of the anesthesia on the maxillary sinus, one of us (W. C. D.) employs it as a routine in dental cases, with good results. The only untoward result has been hematoma with swelling of the cheek.

We have not tried to use this procedure in injecting alcohol into the sphenopalatine ganglion, but it suggests itself as an easy method for this purpose as well as for injection into the second division of the fifth nerve. Other methods may reach the nerve more directly, but the quantity injected in the present method seems to infiltrate the same areas.

1930 Chestnut Street.

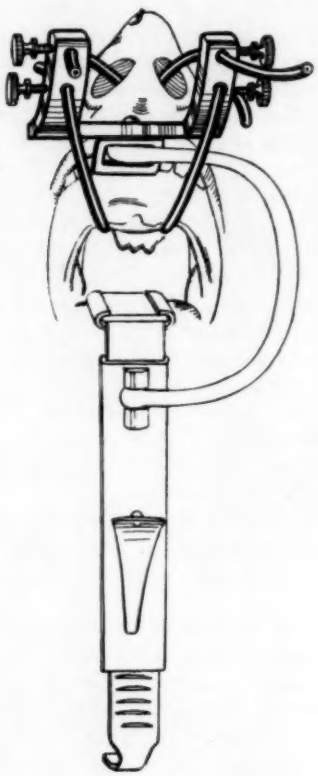
A NEW SOFT PALATE RETRACTOR PERMITTING ADENOIDECTOMY UNDER DIRECT VISION

SIDNEY H. GIDOLL, M.D., SAN FRANCISCO

Recurrence of adenoid lymphoid tissue after previous apparent removal can be attributed to incompleteness of the operation. This may be due not so much to lack of thoroughness on the part of the surgeon as to his inability to do a complete adenoidectomy because of inadequate exposure.

As the adenoid is hidden behind the uvula and soft palate, within the nasopharynx, the operation for its removal must of necessity be done indirectly by palpation rather than under direct vision. Often, too, when the La Force adenotome is used, the uvula may be crushed or pinched, which adds to the postoperative discomfort. Furthermore, it appears that the principles of good surgery require the removal of the adenoid to be done under direct vision, at least for the sake of hemostasis.

It was to overcome these obstacles that the instrument presented here was devised. It retracts the soft palate, exposing the adenoid completely, and does not



require the services of an assistant to depress the tongue. The hands of the surgeon are free for the operation itself, as it is not necessary for him to use one hand to assist in the retraction of the soft palate.

The instrument consists of a modification of the Greene mouth-gag. Utilizing the old principle of a nasal catheter in each nostril, it has the new feature of an attachment to the mouth-gag which anchors the catheters in place by means of thumbscrews. The attachment is constructed at an angle of 45 degrees from the vertical so as to permit easy access to the nostrils or throat.

A no. 12 French catheter (lubricated with sterile liquid petrolatum) is inserted into the upper aperture on one side of the attachment, drawn through and anchored in place by means of its corresponding thumbscrew. The free tip of the catheter

is now inserted through the nostril on the same side, passing posteriorly and downward through the choana into the nasopharynx, where it is picked up by forceps, drawn forward and upward out of the buccal cavity and inserted through the lower aperture of the attachment on the corresponding side, drawn tightly and anchored in place by means of the lower thumbscrew. The same procedure is followed on the opposite side for the other nostril. It is obvious that the tighter the catheters are drawn, the greater will be the retraction upward of the uvula and soft palate and therefore the more complete the exposure of the adenoid mass. Reference to the accompanying diagram will disclose the procedure to be simpler than it sounds.

The aforementioned technic has aided me in producing a resultant clean nasopharynx, as it obviates the necessity of a subsequent operation to stop a secondary hemorrhage or to remove adenoid remnants. Reflex earaches after curettage of the adenoids have been eliminated, since avoidance of the cartilaginous mouths of the eustachian tubes was made possible.

This instrument was made for me by J. J. Huebler & Co. of San Francisco.

516 Sutter Street.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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TONSILS AND ADENOIDS

FRENCH K. HANSEL, M.D.
ST. LOUIS

In reviewing the literature for 1933 on the subject of the tonsils and adenoids, only those papers were selected which promote discussion and which, according to my judgment, show the advances made in this particular field of otolaryngology. Several interesting papers have been published on the relation of the tonsils to general diseases. A few reports on anatomy, physiology, bacteriology and pathology represent further advances in the knowledge of these phases of the subject. It is noteworthy that more interest has been shown in the problem of complications than in any other subject. Further statistics and observations have been reported on the treatment of peritonsillitis and the management of cases with thrombosis of the jugular vein. Further observations have been made on the use of electrosurgical methods of tonsillectomy.

GENERAL CONSIDERATIONS

Kahler [1] discusses the physiologic rôle of the tonsils and the so-called "chronic tonsillitis," particularly in its relation to general diseases. The tonsils belong to the lympho-epithelial system and must be considered as an integral part of the total mesopharyngeal mucous membrane. The lymphatic tissue distributed in the rest of the pharyngeal and intestinal mucous membrane performs the same function as the tonsillar tissue. The hypotheses which consider the tonsils acting as a protective mechanism or functioning as endocrine glands are discussed. Although little is known about the function of the tonsils, they form a very small part of the lymphadenoid tissue of the oral cavity and pharynx. Kahler believes that complete removal of the tonsils does not do any harm, and this is confirmed by clinical experience.

The pathologic findings in chronic tonsillitis and their relation to other diseases, such as rheumatic diseases, endocarditis, septic diseases and nephritis, are discussed. The observations that are made are illustrated by photomicrographs. Tonsillectomy is greatly preferred to any other method of treatment in chronic tonsillitis.

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1. Kahler, Otto: The Tonsil Problem, J. Laryng. & Otol. **48**:2, 1933.

In a clinical lecture by Burger,[2] the origin of angina in relation to the functions and the significance of angina in several conditions are discussed. The author reports cases illustrating alleviation or quick cure of a rheumatic condition of long duration by tonsillectomy; however, this effect could not be verified by statistics. Measures other than tonsillectomy have no value. Frequent attacks of angina are also an indication for tonsillectomy in the period between two attacks. In every case of angina with an infectious complication, tonsillectomy must be considered. Rheumatic conditions that begin with an attack of angina are often not of too long standing for tonsillectomy to be performed with the desired result. The coexistence of rheumatic conditions and chronic tonsillitis is an absolute indication for tonsillectomy. The presence of enlarged lymph glands behind the angulus mandibulae verifies a doubtful diagnosis. Tonsillectomy performed by a skilled surgeon is almost free from danger. In 2,000 tonsillectomies, only 2 patients died (1 death was due to the narcosis and the other to status thymicolymphaticus). Severe hemorrhages were not seen. Only once was the bleeding so severe that a second operation had to be performed.

A monograph of 171 pages by Gording and Bjorn-Hansen [3] is based on exhaustive blood counts and metabolic tests on 137 patients, to decide what symptoms of a general and rheumatic nature may be ascribed to such infective sources as teeth and tonsils, and also how far the Arneth leukocytic shift to the left may be trusted as a diagnostic means of recognizing such focal infective changes.

Removal of foci, with subsequent observation during three years, was carried out on 64 persons with a history of long-standing arthritic changes without acute rheumatic fever. Of this number, 52, with an average age of 31 years, became free from clinical signs and were considerably improved. The blood picture showed infection in 46, the picture returning to normal in 43 persons, while in 3 with pulmonary symptoms the abnormalities persisted.

Twelve persons formed the second, or "acute rheumatic," group, average age 39 years; these patients had a history of acute articular rheumatism. Seven of the 8 patients who were improved after removal of teeth or tonsils showed normal blood at the end; 1, with involvement of the lungs, did not show the improved blood picture. Recurrences were frequent in this group, occurring in 4 of the patients who showed improvement, though recurrences were noted in only 2 of the 52 patients with long-standing arthritic changes but without acute rheumatic fever.

Rather an exact correspondence between a leukocyte count above the normal one of 6,500 with increased shifting to the left and failure

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2. Burger, H.: Tonsils and Rheumatism, Nederl. tijdschr. v. geneesk. **76:** 1451, 1932.

3. Gording, R., and Bjorn-Hansen, H.: Focal Infection of Tonsillar and Dental Origin, Acta Otolaryng., supp. 17, 1933.

to secure relief after removal of foci was observed in both classes of patients. Complete protocols for 64 cases are given. The monograph is excellent and carefully written and presents an accurate analysis of accurately recorded data. A five page summary in English is included.

Horak and Poleduak [4] discuss the results of tonsillectomy in cases of internal disease on the basis of personal observations gathered since 1924. Tonsillectomy was usually performed after regression of the acute tonsillitis, and the acute attack lasted weeks or months without exhibiting any tendency to improve. In cases of sepsis, endocarditis and nephritis, tonsillectomy was undertaken as soon as a relation between the internal disease and the tonsillar disease was ascertained. In cases of chronic disease, tonsillectomy was performed at any stage during a subfebrile period. The best results were obtained in cases of articular rheumatism. Only a small percentage of patients were treated by tonsillectomy, but of those treated, 75 per cent were cured, 23 per cent were improved and only 2 per cent were not improved. In cases of heart disease the results were also good, but 13 per cent of the patients showed no improvement. In the cases of renal disease in which tonsillectomy was performed, focal nephritis occurred most frequently and responded most favorably. Good results were also obtained in cases of sepsis of oral origin. Among the patients with neurologic disturbances who were treated by tonsillectomy, the best response was obtained in those with chorea minor. Comparison of the results obtained by tonsillectomy in internal diseases with those obtained by other forms of treatment showed the therapeutic value of tonsillectomy in internal diseases of a generally septic character. The authors list the contraindications. They point out that if the hematogenic spread of infectious material from the tonsils into the body has been going on for a long time tonsillectomy often cannot arrest the septic process in the body.

Miller [5] points out that during the present century much emphasis has been laid on chronic infection of the tonsils as a source of constitutional ill health and systemic disease, and hence the tendency to wholesale or indiscriminate removal of tonsils. The influence of septic tonsils and the value of removal in certain conditions are discussed in detail.

The general picture of subnormal health in children in whom are seen loss of weight, appetite and color and the development of excessive fatigue, irritability and nervousness may be greatly benefited by the removal of infected tonsils. Miller stresses that failure to obtain complete and permanent return to good health after tonsillectomy may be due to chronic intestinal toxemia based on the primary tonsillar sepsis.

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4. Horak, J., and Poleduak, L.: Tonsillectomy in Internal Diseases, Ztschr. f. Laryng., Rhin., Otol. **23**:135, 1932.

5. Miller, Reginald: Some Aspects of Tonsillitis and Tonsillectomy in General Medicine, Practitioner **129**:521, 1932.

With regard to colds and catarrhal conditions of the nose and throat, Miller believes that tonsillectomized children are much better protected than those with septic tonsils, but there is no reason to believe they are better protected than children who have healthy tonsils. The value of tonsillectomy in asthma and juvenile rheumatism is also presented.

Eigler [6] says that a study of the cases of tonsillitis at his clinic during one year showed that the number of cases increased during the months of seasonal change, during changes in weather, during periods of constant high atmospheric pressure associated with a drop in temperature and during periods of low atmospheric pressure. Strong wind and storms also seemed to favor the development of this condition. About one half of the cases of tonsillitis appeared after operations on the upper respiratory tract, especially after septum resection. In 57 cases in which submucous resection was performed, bacteriologic examinations showed a decided change in the flora of the pharynx and of the tonsils several days after the operation; the number of hemolytic streptococci in the epipharynx and the tonsils was greatly increased, and they were usually found abundantly in pure culture in the nose. Not all patients with hemolytic streptococci who were operated on had tonsillitis, but hemolytic streptococci were found in all patients in whom postoperative tonsillitis developed. In almost all of 10 cases in which the opsonic index of the blood was determined, the patients showed an increased opsonic index for their own streptococci several days after the tonsillitis receded. These facts indicate that the streptococcus is the etiologic agent in postoperative tonsillitis. The epithelial cells of the tonsils and pharynx appear to have a phagocytic function; those of the nose do not. Examination of the pharyngeal mucosa before, during and after inflammation showed, besides the normal conditions, pharyngitis granularis, diffuse cellular inflammation, edema and inflammatory fibrous hyperplastic conditions. The author concludes that the atmospheric conditions, the hemolytic streptococci and the existing condition of the pharyngeal mucosa before intervention are the determining factors in postoperative tonsillitis.

Hindes and Powschitkow [7] call attention to chronic tonsillitis as a source of postoperative complications. They conducted necropsies in 7 of 14 cases of unexplained postoperative death which occurred following 10,256 gynecologic operations. In 4 of these 7 patients suppurating foci were found in the tonsils which must be regarded as the source of the septic peritonitis and septicemia which caused death. In the other 3, the origin of the septic peritonitis could not be ascertained. The

6. Eigler, G.: Tonsillitis and Weather, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:119, 1933.

7. Hindes, M. E., and Powschitkow, W. A.: Chronic Tonsillitis as the Etiologic Factor in Postoperative Complication in Gynecology, Monatschr. f. Ohrenh. **66**:1427, 1932.

authors think that all patients should be examined prior to operation, in cooperation with the laryngologist and the internist. When chronic tonsillitis is present in a patient requiring surgical intervention, healing of the diseased tonsils should be effected first. In all cases of postoperative death of unexplained origin, postmortem examination of the tonsils is recommended.

Kindler [8] differentiates between the pure form of agranulocytosis, which is of unknown cause, and the symptomatic form, which has a known toxic or infectious basis and leads to a syndrome of agranulocytosis. He reports a personally observed case of symptomatic agranulocytosis associated with septicemia of tonsillar origin. The etiologic diagnosis was complicated by the fact that the patient had just received a course of antisyphilitic treatment and that either the syphilis or the neoarsphenamine might have caused the agranulocytic syndrome. Tonsillectomy and drainage of a retrotonsillar abscess resulted in an immediate disappearance of the necroses and improvement of the clinical symptoms; the agranulocytic changes in the blood persisted for several months before gradually receding. The author thinks that the neoarsphenamine therapy, through injury to the leukopoietic system, paved the way for the agranulocytosis. The occurrence of severe pneumococcic tonsillitis with abscess formation caused the clinical manifestations of the agranulocytosis in the form of necroses of the mucosa associated with a picture of severe sepsis.

In a report on the subject of so-called thymic death by Waldbott,[9] consideration is given to respiratory sensitization to local anesthetics. The author points out that many cases of so-called thymic death are not a result of any changes in the chest but represent a form of fatal anaphylaxis. He briefly outlines some of the measures for the prevention of this complication when administering anesthetics.

According to Brown, in Queensland, Australia, affections of the nose and throat are more common than in Great Britain, and the removal of tonsils and adenoids is indicated in a great many cases. The frequency of diphtheria carriers and persons with disease of the mastoids among patients with tonsils and adenoids is discussed. The statistics on cases observed in the Hospital for Sick Children during a period of seven years are given.

Brown [10] found that diphtheria was rare in children who had had their tonsils completely removed, and patients who had been diphtheria carriers were declared free from the disease in an average time of ten

8. Kindler, W.: A Case of Agranulocytosis Cured by Tonsillectomy, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:85, 1933.

9. Waldbott, George L.: So-Called Thymic Death, Arch. Otolaryng. **17**:549, (April) 1933.

10. Brown, R. G.: The Relation of Diphtheria and Diphtheria Carriers and Mastoid Disease to Tonsils and Adenoids, J. Laryng. & Otol. **47**:673, 1932.

days after tonsillectomy. He believes that the incidence of middle ear and mastoid diseases has greatly decreased within the last few years, and he attributes the decrease to the greater frequency of operations for removal of tonsils and adenoids.

Keen [11] discusses certain abnormal conditions and special problems encountered in a series of 10,000 cases of operations on the tonsils and adenoids. A prominent tubercle of the atlas was found in 1 case in every 400. This condition may cause difficulty in the removal of the adenoids, especially if one uses the curet. The styloid process could be felt in the empty tonsil fossae in 1 case in every 5,000. In 2 instances an unexpected choanal polypus was found at operation, and in 3, a latent peritonsillar abscess, but in none of the cases were there any complications following the operation.

The problem as to whether in certain cases the tonsils should be removed as well as the adenoids is discussed. Operations on the turbinates should be avoided as far as possible in children. The author does not approve of the finger examination for adenoids in children. The La Force adenotome is greatly to be preferred for the removal of adenoids to the adenoid curet.

In an article dealing exclusively with adenoids, Pierce [12] describes carefully their embryologic development and anatomy. In regard to function he states that they act as a test organ for respiratory fluids, i. e., the inspired air current and the systemic blood. In this capacity they take samples of all atmospheric micro-organisms and also prepare immunizing substances. Another function attributed to adenoids is the manufacture of hormones in connection with the endocrine system.

In the etiology of hypertrophy, Sturm places heredity as a predominating factor in about 50 per cent of cases. Syphilis, tuberculosis and rickets apparently are not causal factors. Cases are distributed equally among breast-fed and bottle-fed children, and also between the sexes. The author believes that, apart from heredity, the predisposing factors of improper food and foul air are of great importance. In regard to the relative frequency of hypertrophied adenoids and hypertrophied tonsils, in a series of 572 patients operated on by the author, 15 had enlarged tonsils only, 282 had enlarged adenoids only, and in 275 both were hypertrophied. The treatment recommended is surgical removal, and the author, after using the La Force adenotome in more than 3,000 consecutive cases, believes that it is the most efficient instrument available. A detailed description of the technic, including diagrams, is given.

Lemere [13] reports further observations on the adenoids and immunity. The conclusion was drawn in his previous paper that the adenoid

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11. Keen, J. A.: Some Observations on the Tonsil and Adenoid Operation in Children, J. Laryng. & Otol. **47**:449, 1932.

12. Pierce, F.: Adenoids, Practitioner **129**:500, 1932.

13. Lemere, Henry B.: Adenoids and Immunity, Arch. Otolaryng. **18**:326 (Sept.) 1933.

is a physiologic lymphatic structure admirably designed to expose the greatest possible lymphatic surface as a barrier to the entrance of nasal infection through the nasopharyngeal mucosa into the general circulation.

In a series of more than 300 children, the adenoids were left at the time the tonsils were removed. He summarizes the results of treatment in relation to mouth breathing, acute otitis media, catarrhal deafness, colds, broad nasal bridge and disease of the sinuses.

A group of 179 adults was observed by Gafafer [14] for thirty-five weeks from Sept. 29, 1929, to May 31, 1930, and every effort was made to secure reports of all attacks of disease of the upper respiratory tract (common cold). Of these adults, 123 had tonsils and adenoids and 56 did not. The group with tonsils and adenoids and the group without tonsils and adenoids presented no significant difference with respect to frequency, severity or type of attack of disease of the upper respiratory tract (common cold).

According to Orosz and Kugler,[15] of 161 children on whom the Schick test was performed from one to six years after they had had their tonsils and adenoids removed, 80.7 per cent of those between 2 and 6 years of age, and 68.7 per cent of those between 6 and 12 years of age, responded with a positive reaction. The results were the same in children in whom hyperplasia of lymphoid tissue had occurred following tonsillectomy and adenoidectomy. The authors found no relation between tonsillectomy and the Schick reaction, and hence were unable to confirm the observations of Schick and Topper published in November 1929.

Topper and Leader [16] state that there is considerable clinical evidence that tonsillectomy favors the development of immunity to diphtheria. It is probable that the development of immunity after tonsillectomy depends largely on the presence of diphtheria bacilli in the environment and is therefore most likely to occur in congested areas in which the incidence of diphtheria is high. The authors report the results in 81 children who gave positive reactions to diphtheria before tonsillectomy and who returned for retesting six months later. The reactions of 27 (33.3 per cent) remained positive, and those of 54 (66.6 per cent) became positive. Among those whose reactions remained positive were 3 children who had received toxin-antitoxin several years before tonsillectomy; 2 of them had received two series of prophylactic inoculations. The number of susceptible children, as shown by the Schick test, who

14. Gafafer, W. M.: Adenotonsillectomy and Common Cold in Adults, J. Infect. Dis. **51**:489, 1932.

15. Orosz, D., and Kugler, G.: Tonsillectomy and the Schick Reaction, Arch. f. Kinderh. **94**:168, 1931.

16. Topper, Ann, and Leader, S.: Effect of Tonsillectomy and Adenoidectomy on Immunity to Diphtheria, Am. J. Dis. Child. **45**:735 (April) 1933.

acquired immunity after tonsillectomy was smaller than the number reported by Schick and Topper.

In the study on the effect of tonsillectomy on the development of immunity to scarlet fever as shown by the Dick test, Kereszturi and Park [17] state that, from their limited number of cases, it may be said that tonsillectomy has certainly no marked effect on the susceptibility to scarlet fever within six months after it is done, as demonstrated by the changing of a positive Dick reaction to a negative one.

Twenty cases of septic sore throat reported by Archibald and Feemster [18] were definitely traced to milk from a single cow. The rate of infection was high, 87 per cent of the persons definitely known to have used the milk raw becoming ill. There were 3 deaths, giving a fatality rate of 15 per cent. Two patients died of pneumonia, and 1 of otitis media, cellulitis and septicemia. Hemolytic streptococci were found in cultures taken from the throats of 15 of the patients and from that of the milker, and in milk from the diseased quarter of the cow's udder. No cases developed in contacts in the family not drinking the infected milk.

The usual clinical picture was that of a sudden onset, with chilliness, fever and a dry, raw throat. There were also nausea, vomiting, backache and prostration. Examination of the throat showed a bright red pharynx, uvula and soft palate, no exudate being visible. In all of the cases there were more or less swelling and tenderness of the cervical glands.

ANATOMY AND PHYSIOLOGY

Sasaki [19] says that careful histologic investigation shows that the indentation of the tonsils usually called the "fossa supratonsillaris" is always underneath the tonsillar capsule and has the same structure as the tonsillar crypts. As the term "fossa" is superior to "capsule," the author suggests substituting the term "fossa tonsillaris superior." The fact that the fossa is found in the tonsillar parenchyma has a clinical significance, and its structure must be especially considered in enucleation of the tonsil. The structure of this fossa also probably plays a large rôle in recurrent tonsillitis and peritonsillitis and their complications.

Brunner and Schnierer [20] studied the arterial supply of the tonsils at necropsy in 6 specimens and found a much greater variation than

17. Kereszturi, Camille, and Park, William H.: Effect of Tonsillectomy on the Development of Immunity to Scarlet Fever, J. A. M. A. **101**:764 (Sept. 12) 1933.

18. Archibald, Robert E., and Feemster, Roy F.: A Small Milk-Borne Outbreak of Septic Sore Throat, New England J. Med. **206**:1252, 1932.

19. Sasaki, M.: Fossa Supratonsillaris, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:89, 1933.

20. Brunner, H., and Schnierer, J.: Arteries of the Palatine Tonsil, Monatschr. f. Ohrenh. **66**:1180, 1932.

was found by Lund in his examination of 20 specimens. They agree with the latter author that the arteries usually pierce the superior pharyngeal constrictor at the level of the lower pole of the tonsil and enter the capsule at this level; but, contrary to Lund's observations, they also found arteries in the upper pole of the tonsils in cases in which the tonsils were supplied by several arterial branches. In the new-born they found that the arterial branch pierces the constrictor and penetrates from the side through the capsule into the tonsil. The arteries may traverse the parenchyma and ramify on the medial surface of the tonsil, thereby penetrating into the fold of mucous membrane which covers the supratonsillar fossa. In adults they found the capsule artery, described by Lund, only once, and even in this case there was another artery which appeared under the tonsil and extended to the front to supply the inferior pole of the tonsil and the anterior palatine arch. In 2 other cases the arterial branches tended to encircle the tonsil and then penetrate the parenchyma from the front or the back, also supplying, respectively, the anterior or the posterior palatine arch. So far as the derivation of the arteries is concerned, they found that in 2 cases (in a fetus and in a new-born infant) the tonsil was supplied by the ascending palatine artery, which originates in the external maxillary artery; in 2 cases, by the ascending palatine and the external carotid, and in 1 case by two branches of the external maxillary artery as well as by the external carotid. These authors agree with Lund that the most important vessels are in the lower pole of the tonsil and that arterial hemorrhage is most to be feared in this region. They consider it surprising that postoperative arterial hemorrhages are not more frequent under these variable conditions. They attribute the postoperative closing of the tonsillar arteries to their strong annular musculature and the intima cushions of the tonsillar arteries, described by Brunner.

Brunner [21] discusses the structure of the blood vessels of the tonsillar capsule on the basis of personal examination of many specimens. The arterial vessels of the tonsil are characterized by an unusually thick tunica elastica interna which can be demonstrated in the walls of their finest ramifications. In the larger capsular arteries, intima cushions which do not show a uniform structure are found, frequently in adults and rarely in children. In surgically obtained tonsils one frequently finds artefacts in the larger capsular arteries caused by rupture of the intima and media. The larger capsular veins may be divided into those rich in muscles and those poor in muscles. All these peculiarities in the structure of the capsular vessels are functionally related to the large variations of the blood supply and circulation within the tonsil. The variations in the blood supply of the tonsil are in turn related to the

21. Brunner, H.: Structure of Arteries and Veins in the Tonsillar Capsule, Monatschr. f. Ohrenh. **66**:1335, 1932.

fluctuating occurrence of the secondary nodules and to the growth and involution of the tonsils. Occasionally in the capsule completely thrombosed veins are seen, in the vicinity of which are signs of tissue irritation; in the author's opinion it is doubtful whether or not septic processes originate from these thrombi. Remnants of obturating thrombi are frequently found in large veins in the form of bands of connective tissue traversing the lumen.

In the search for a method of determining whether a given general disease is related to the tonsils, Wirth [22] experimented with intracutaneous injections of tonsillar extracts. He found that intracutaneous injections of tonsillar extracts produced early and late reactions of varying degrees. Some persons are hypersensitive to certain tonsillar extracts. No relations of diagnostic value could be found to exist between the intracutaneous reactions and the clinical picture.

Bischoff and Weinand [23] report the chemical extraction of acetylcholine from human tonsils. The extract was found to have a strong reducing effect on the blood pressure of cats. Since Bischoff, Grab and Kapfhammer have shown that acetylcholine is contained in many other organs, the authors think that the mere presence of this blood pressure-reducing substance in the tonsils does not justify designating the latter as glands of internal secretion.

Mies [24] states that two main theories concerning the etiology of traumatic tonsillitis (tonsillitis after nasal operations) are presented in the literature. One regards traumatic tonsillitis as a secondary infection of the tonsils transmitted from the nose via the lymph channels; the other considers it as a primary surface infection of the tonsils determined by certain factors created by nasal tamponade. In general, a lymphatic connection between the nose and the tonsils is denied since the negative results of Schlemmer's experiments in 1921. Mies conducted experiments on guinea-pigs to determine whether in the pathologic conditions produced by nasal tamponade the flow of lymph was altered. By unilateral nasal tamponade the disturbance of nasal breathing necessary for the occurrence of traumatic tonsillitis was produced. Following the injections of a dye into the nasal mucosa under these conditions, no dye could be demonstrated in the tonsillar tissue. In simple experimental injections into the mucosa of the nose and palate it was likewise impossible to detect any deposits of dye in the tonsils. This corroborates the results of Schlemmer. In both experimental series, particles of dye were sometimes found in the retrotonsillar tissue

22. Wirth, Erich: Intracutaneous Reactions with Tonsillar Extracts, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **134**:4, 1933.

23. Bischoff, C., and Weinand, K.: Blood Pressure-Reducing Effect of Tonsil Extract, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:647, 1932.

24. Mies, C.: Experimental Study of Traumatic Tonsillitis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:52, 1933.

on the side into which injection had been made. The results of animal experiments do not support the assumption of a lymphatic connection between the nose and the tonsils. The Denker theory of the etiology of traumatic tonsillitis, which assumes a secondary infection of the tonsils from the nose via the blood stream, appears to be the most plausible and well founded theory.

On the basis of an extensive experimental investigation on human beings and on animals, Waldapfel [25] states that substances introduced into the mucosa of the inferior nasal concha reappear in the tonsils, where they can be demonstrated microscopically and to some extent chemically; substances introduced into other parts of the nasal mucosa are conveyed exclusively to the regional lymph nodes. Organic material reaches the tonsils in larger amounts than inorganic material, cellular material in still larger amounts and bacteria most abundantly. The coloring matter is distributed in varying amounts in different parts of the tonsil. The substances are conveyed from the nose into the tonsil by the efferent veins of the cavernous plexus of the inferior conchae which communicate with the veins of the pharynx, which in turn communicate with the tonsils. As the veins of the two sides of the pharynx communicate with each other, the substances injected unilaterally into the concha appear in both tonsils. Following intravenous injections in other parts of the body, a small part of the injected material reaches the tonsil via the blood stream, but it is a much smaller proportion than that carried by the venous route from the cavernous plexuses of the nose. The substances which reach the tonsils are eliminated into the crypts through the epithelium, usually where it is reticulated but also through intact portions. From six to twelve hours after intravenous injection the injected substance can be demonstrated in the crypts. Only small particles of bacterial size are eliminated. A part of the material entering the tonsil is conveyed to the regional lymph nodes. These experimental results confirm the opinion of those who attribute to the tonsillar tissue the function of ridding the blood stream of some of the foreign material which has entered it. They also attest that infection can spread from the nose to the tonsils, not via the lymph channels as is usually assumed, but by the venous route. This is possible because of the cavernous plexuses of the nasal conchae which bring the venous blood so close to the exposed surface that infectious agents can enter it following the slightest injury of the protective covering.

Full-grown rabbits were used in an investigation of the function of the tonsils by Scherf.[26] When colloidal trypan blue or silver or finely

25. Waldapfel, R.: Experimental Histologic Studies of the Tonsil, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:400, 1932.

26. Scherf, H.: Function of the Tonsils, Virchows Arch. f. path. Anat. **283**:74, 1932.

particulate india ink was painted on the surface of the tonsils, the material was not taken up by the lymphoid tissue. Spraying of the same substances into the pharynx led to deposition of the material on the surface epithelium, but it was not transferred to the tonsil itself. The surface of the tonsil may mechanically remove foreign particulate matter from inspired air. When the experimental materials were injected into the submucous tissues of the nasopharynx and mouth, they were stored in macrophages of the peritonsillar tissue, but did not reach the tonsillar tissue itself, indicating that peritonsillar inflammatory processes may result from lymphatic transport from the submucous tissues. When the materials were injected into the common carotid artery, the material was stored in the capillary endothelium, from which it was passed to the surrounding tissue. This process was most marked in the subepithelial capillaries, the material passing to the surface of the tonsil and being excreted. The tonsils may apparently function in the removal of foreign material from the blood stream.

Kelemen and Hassko's [27] study of the absorption of colloidal silver in the tonsils constitutes the third series of experiments in an investigation of the absorption of foreign matter in the tonsils. In the present series, the authors experimented with collargol and electrocollargol on human beings and rabbits. With a single exception, no silver could be histologically demonstrated in the tonsillar tissue after application of collargol to the surface of human tonsils; neither could silver be demonstrated in the tonsils after intravenous injections of customary doses of electrocollargol. Collargol and electrocollargol injected intravenously into rabbits in the doses they tolerated did not reach the tonsils; only when lethal doses were injected could traces of silver in the tonsils be demonstrated. With injection of collargol after elimination of the reticulo-endothelium of the spleen and liver by colloidal copper (amounts up to the lethal dose), the tonsils also remained free from silver even when conditions for phagocytosis were improved by gelatin. Likewise, silver injected into animals after splenectomy and blockade of the liver by copper, and silver injected into a human being after splenectomy, could not be demonstrated in the tonsils of the experimental subjects. The authors conclude that the tonsil does not possess any elements capable of absorbing significant amounts of silver. Even in severe generalized argyrosis there is no significant absorption of silver in the tonsils as there is in the tissue of the surrounding organs.

PATHOLOGY AND BACTERIOLOGY

Pilot and Rosenblum [28] made a bacteriologic study of infections of the throat in children during the winter and spring of 1931, from

27. Kelemen, G., and Hassko, A.: Absorption of Foreign Matter in Tonsils: Tonsils and Colloidal Silver, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:326, 1932.

28. Pilot, I., and Rosenblum, P.: Sore Throat in Children Due to Streptococcus Epidemicus, Am. J. Dis. Child. **44**:994 (Nov.) 1932.

January to May. They found that 9 of 102 children with sore throat yielded Streptococcus epidemicus; 50 others yielded ordinary hemolytic streptococci. The sore throat due to Str. epidemicus was sporadic and not related to the supply of milk. The clinical picture of the patients with tonsillitis due to Str. epidemicus was similar at the onset to that of the patients with ordinary hemolytic streptococcic infections, but complications and sequelae were more common in the former. The greater frequency of complications may be due to the capsules and the mucoid character of Str. epidemicus, properties that have been associated with added virulence and aggressiveness.

By the injection of staphylococci and streptococci into the carotid artery of rabbits and cats, Krauspe [29] found that an acute, diffuse inflammation of the tonsils was set up. This reaction was often as superficial as that of the spontaneous necrotizing tonsillitis of rabbits, which is due to infection from the surface of the tonsil. When rabbits were sensitized by repeated injections of streptococci subcutaneously, injection of the same strain into the carotid artery caused lesions that were more circumscribed than those caused by the same organism in nonsensitized animals. Such lesions consisted chiefly of histiocytes. Similar small granulomatous lesions were seen also in the tongue, myocardium and kidney. The injection into the carotid artery of bacteria-free, toxin-containing filtrates of streptococci caused inflammatory changes in the tonsils.

Krauspe [30] examined the palatine tonsils of 11 children and 14 adults who had died as the result of generalized bacterial infections. In the majority of cases, especially in the children, hematogenic involvement of the tonsils was demonstrable. Streptococcic infection was predominating in the children, and in these cases extensive inflammatory changes were noted, particularly in the blood vessels of the follicles and in the subepithelial capillaries. The changes showed all degrees of severity, from slight perivascular inflammation to extensive suppuration and necrosis. In severe cases there existed follicular abscesses and necrotic suppurative inflammations of the crypts. The primary stages generally showed the aspects of resorptive, catarrhal or ulcerous infections. Demonstration of the bacteria was usually possible without difficulty. Most of the bacteria were phagocytosed. The subepithelial tissue showed that the bacteria had entered the crypts through the epithelium. In the course of staphylococcic infections in adults, suppurative inflammations of the crypts and abscesses of the follicles were frequently found in the palatine tonsils. Acute streptococcic infections produced

29. Krauspe, C.: Experimental Hematogenous Tonsillitis, Virchows Arch. f. path. Anat. **285**:400, 1932.

30. Krauspe, C.: Changes in Palatine Tonsils in Course of General Infection: "Hematogenous Tonsillitis," Virchows Arch. f. path. Anat. **287**:139, 1932.

in the tonsils of adults changes similar to those observed in children, and some of these changes had the aspect of primary infections. A metastatic involvement of the tonsils was also observed in a case of typhoid fever and in one of glomerular nephritis. In the case of glomerular nephritis, the tonsillar involvement had the aspect of peculiar focus-like necrosis of the vascular loops and of swelling of the ground tissue, particularly in the follicles. On the basis of these observations the author concludes that hematogenic involvement of the palatine tonsils is much more frequent than was formerly believed and that differentiation of resorptive from hematogenic tonsillitis is extremely difficult, because of the great similarity in the anatomic aspects.

It is well known that sometimes after tonsillectomy there are recurrences of previously existing systemic diseases, such as polyarthritis, nephritis or endocarditis. The explanation of this observation by some writers is that pathogenic organisms and toxins might enter the circulation in connection with the removal of the tonsils. This contamination of the blood stream occurs more often if the tonsils are acutely inflamed before removal.

Wirth [31] made five cultures of blood for each of 45 patients before and after removing their tonsils. Fifteen did not have acute inflammation of the tonsils at the time of operation. Five had recovered from an acute attack from one to three weeks prior to the operation. In 20 cases, besides recurrent sore throat, general systemic disease was present. In all of the 45 patients the five cultures of the blood were negative.

The tissue reactions that Klinge [32] has described as characteristic of rheumatic infection occur in collagenous connective tissue. Such changes are therefore not to be expected in the reticulo-endothelioid lymphoid tissue of the tonsil itself. But in necropsy material rheumatic lesions have been observed in the peritonsillar tissues. Examination of tonsils removed during the acute or subacute stage of rheumatic fever has yielded inconstant results. In 2 pairs of such tonsils examined by Klinge, there were numerous rheumatic lesions of the peritonsillar tissue, and in a third pair there were a few lesions, but in other tonsils the findings were negative. The results of examination of tonsils removed during a first or a recurrent attack of rheumatic fever, taken in connection with the results of Sarafoff's examination of tonsils and tissues of the neck at necropsy, warrant the assumption that the rheumatic virus may enter the body through any part of the upper respiratory tract as well as through the tonsils.

The histologic examination of the pharyngeal tissues and cervical lymph nodes of 4 patients who died of rheumatic fever uncomplicated

31. Wirth, E.: Do Pathogenic Micro-Organisms Get into the Circulation During Tonsillectomy? Deutsche med. Wchnschr. **58**:1755, 1932.

32. Klinge, F.: Tonsillar Changes in Rheumatic Fever, Virchows Arch. f. path. Anat. **286**:333, 1932.

by any other disease is reported by Fraser.[33] Two patients (a man aged 20 and a boy aged 8 years) had had recurrent attacks of rheumatic fever. The other 2 patients (boys, aged 8 and 7 years) died during the first attack.

Rheumatic nodules were found in the pharyngeal tissues. These nodules were more commonly found in the vicinity of the tonsils, radiating from the tonsillar capsule into the connective tissue of the muscles of the pharynx.

Similar lesions were present in the region of the lingual and laryngeal tonsils in one of the recurrent cases. In this case there were also significant changes in the regional lymph nodes. These changes consisted of marked endothelial hyperplasia with much polymorphonuclear and plasma cell infiltration. Aschoff nodules were found in the connective tissue surrounding the vessels in the capsule and fibrous stroma of some of the nodes in the upper, deep cervical group on both sides.

In connection with a case of primary tuberculosis of the tonsils, confirmed by necropsy, in a nursling aged 3 months, Hamperl and Wallis [34] point out the difficulty of the diagnosis of this condition from the anatomicopathologic and especially the clinical standpoint. Cases in which an unquestionable diagnosis of primary tuberculosis of the tonsils can be made are extremely rare. In the case described, the mother of the child had active pulmonary tuberculosis, with many acid-fast bacilli in the sputum. It is probable that the transmission of the tubercle bacilli occurred through the mother's practice of moistening the nipple of the nursing bottle with her own saliva.

Rossner [35] states that tuberculosis of the tonsils is probably always a secondary infection, originating by the hematogenic or lymphogenous route or in the sputum. The hematogenic route is the most probable in the opinion of the majority of authors. Tonsillar tuberculosis usually assumes a light form, and in the majority of cases it can be recognized only microscopically. Complications following the removal of tuberculous tonsils were rarely seen; in only a few cases was the operation followed by miliary tuberculosis. In the author's clinic, tuberculosis was found in 3 of 296 tonsils examined histologically, or 1.01 per cent. The statistics in the medical literature vary between 5 and 1.6 per cent.

Gerstel [36] reports an unusual condition first described in 1873 by E. Frankel under the name "benign lingual and tonsillar mycosis."

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33. Fraser, A. D.: Lesions of the Pharynx in Acute Rheumatism, Arch. Dis. Childhood **7**:181, 1932.

34. Hamperl, H., and Wallis, K.: Primary Tuberculosis of the Tonsils, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:480, 1933.

35. Rossner, Therese: Frequency and Significance of Tuberculosis of the Tonsils, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:214, 1933.

36. Gerstel, G.: A Case of Papillary Hyperkeratosis of Waldeyer's Tonsillar Ring, Virchows Arch. f. path. Anat. **286**:116, 1932.

Death was due to pulmonary tuberculosis in Gerstel's case. On the base of the tongue and in the pharynx, in the region of the lingual, faucial and pharyngeal tonsils that make up Waldeyer's ring, were numerous elevated, rounded and thornlike hyperkeratoses. These arose from the squamous epithelium of the crypts of the lymphoid tissue. The author disagrees with the view of some observers that the condition is a mycosis. No fungi could be seen in the lesions, and the bacteria present were held to be secondary invaders. The process is not inflammatory. It is an epithelial hyperplasia that is sharply limited to the crypts of lymphoid tissue, where multiple localized areas of leukoplakia most often occur.

Fraenkel [37] reports a case of cavernous angioma of the left tonsil in a man, aged 29. Only 7 other cases of this condition have been reported. There was an anamnesis of frequent tonsillitis and occasional hemorrhages from the throat after violent coughing. The left tonsil was greatly enlarged. It was dark red and nodular, resembling a blood-soaked sponge. A diagnosis of tonsillar angioma was made, and the tonsil was extirpated, together with the adherent glossopalatine arch, after ligation of the plainly visible vessels. The tumor was 5 by 2.5 cm., dark brown and rough. The cut surface exhibited a honeycomb structure. A nodule of tonsillar tissue 0.5 cm. in diameter was found in the center, as well as a few nodules the size of a pinhead; microscopically, these nodules appeared encapsulated in a thin layer of connective tissue. In place of the reticulo-adenoid tissue usually found in the tunica propria, there was cavernoma tissue consisting of a coarse reticulum of trabeculae surrounding alveolae filled with blood. The author describes the histologic picture in detail.

Benign tumors of the tonsil with special reference to fibroma are discussed by Hara.[38] He gives an interesting review of the literature, with tabulations, and reports an interesting case.

COMPLICATIONS

Claus [39] discusses septicemia following tonsillitis, particularly in regard to diagnosis and therapy. After blood has been taken for bacteriologic and cytologic examinations, a careful anamnesis obtained and examination of the larynx and pharynx completed, the exterior of the neck should be carefully examined. Sensitivity to pressure in the angle of the jaw is significant if there are no acute manifestations in the ton-

37. Fraenkel, I.: Cavernous Angioma of the Tonsil, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:268, 1933.

38. Hara, H. J.: Benign Tumors of the Tonsil, Arch. Otolaryng. **18**:62 (July) 1933.

39. Claus, H.: Septicemia Following Tonsillitis, Ztschr. f. Laryng., Rhin., Otol. **23**:58, 1932.

sils. Cord formations are found in phlegmons and glandular infection and are not a septic symptom unless the blood vessels or the mediastinum is involved. Careful study of the general condition is valuable. Special examination of the lungs, heart and kidneys to determine metastases and of the eyes for symptoms of cavernous sinus thrombosis should be made. Thrombophlebitis of the jugular vein toward the sigmoid sinus may occur. Exposure of the sinus and removal of the thrombus may save the life of the patient. The author thinks that there is always a phlegmonous process in the loose connective tissue of the parapharyngeal space, which spreads to the near or distant lymph vessels or, more often, to the veins, and that the infection never spreads directly via the lymph or blood vessels. The phlegmon spreads toward the mediastinum more often than toward the cranial cavity. Surgical intervention for inspection of the large vessels should be undertaken whenever possible and should consist in an incision along the inner edge of the sternocleidomastoid muscle from the tip of the mastoid process to the clavicle. If the infiltration is severe and orientation is difficult, cervical mediastinotomy is indicated to find the veins; they may then be traced upward to the area of infiltration. Diseased vessels should be resected, whenever possible, following ligation. The healthy jugular vein should be ligated when the facial vein is diseased and in doubtful cases. Elimination of the actual septic focus is important. The author supplements control of the blood vessels with tonsillectomy, since a focus of suppuration is often found behind the normal-appearing tonsil. Enucleation of an acutely inflamed tonsil need not be shunned if the interstitial spaces of the connective tissue have been previously exposed from the exterior and no injection is made into the infected area. Metastases in the lung are not a contraindication to intervention.

Uffenorde [40] discusses the hematogenic lymphogenous and phlegmonous forms of pathogenesis of septicemia following tonsillitis. He thinks that the phlegmonous origin is most common. The infection originating in the tonsils is spread by a progressive phlegmonous process in the interstitial spaces of the neck, which extend into the parapharyngeal space and sometimes to the exterior. The process may extend to the cerebrum and also, but more rarely, to the mediastinum. Periphlebitis in one or several places, near or far from the primary focus, leads to phlebitis and thrombophlebitis. The author thinks that thrombosis of the smallest veins in the tonsillar region has no rôle in the pathogenesis of septicemia. It may be considered more as a protection against the spread of infection than as an endophlebitic septic focus. The various forms of pathogenesis frequently overlap each other. The author empha-

40. Uffenorde, W.: Septicemia Following Tonsillitis, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:41, 1932.

sizes that the tonsil may constitute the septic focus as well as the portal of entry of the infection. Diagnosis of septicemia is often difficult. Chills are sometimes lacking in septicemia, and, on the other hand, they sometimes mark the beginning of some other disease. Careful anamnesis and bacteriologic examination of the blood are valuable. Cytologic examination of the blood is important, not only for differentiation from leukemia, agranulocytosis and tonsillitis with the lymphoid cell reaction, but for control of the blood picture in dubious cases. If the tonsil is the main septic focus, enucleation suffices even if the peritonsillar tissue is also inflamed. In cases in which the spread is lymphogenous or phlegmonous, intervention should be from the exterior and should consist in the thorough airing and draining of all affected interstitial spaces and the parapharyngeal space up to the tonsil and a careful examination of the blood vessels, including the jugular vein and the common facial vein with its branches. Ligation of the jugular vein is not an absolute safeguard against spread of the infection. In case the veins are pathologically altered, the diseased part should be resected after ligation. Ligation of a healthy vein is useless.

Rubin [41] points out that pyemia occurs following acute tonsillar infections in from 1 to 2 per cent of the cases. Chills occurring from two to three days after the beginning of a sore throat, or one occurring a week or two after an angina that has subsided, with a swelling along the anterior part of the sternocleidomastoid muscle or over the parotid region, may be the beginning of pyemia. One must exclude pneumonia, pyelitis or one of the blood dyscrasias, such as agranulocytosis or leukemia. Once the diagnosis is established, immediate exploration of the neck is advisable. When the cavernous sinus is involved, the infection spreads by way of the pterygoid plexus or the petrosal sinuses. It has been demonstrated that thrombi are present in the tonsillar veins in nearly all cases of acute tonsillar or peritonsillar inflammation. It is possible that, in some cases, abscesses of the lungs occurring after tonsillectomy may be explained on the basis that a tonsillectomy is done before the inflammation of the tonsil has subsided. The trauma resulting from the operation causes the phlebitic process to spread. In many cases the inflammation in the tonsil may not be demonstrable. The anaerobic streptococcus is the organism most frequently found. Blood cultures are positive in about 50 per cent of the cases. The presence of anaerobic streptococci or bacilli in the blood stream should make one consider the possibility of a preexisting tonsillar infection as the cause. In two of the author's 4 cases, which he reports, the blood culture was positive. The prognosis appears to be more grave when such organisms

41. Rubin, H.: Pyemia Following Acute Tonsillar Infections, Arch. Otolaryng. **17**:183 (Feb.) 1933.

are present. Clause suggests an exposure of the structures of the neck from the mastoid process down to the clavicle. The parapharyngeal space should be exposed, and any infection found should be dealt with. Ligation or resection of the jugular and facial veins is also carried out. If it is impossible to determine on which side the infection is taking place, he suggests exposing the neck on both sides, and if the jugular vein is involved on one side, he suggests ligating or resecting it and ligating and resecting the facial vein on the opposite side. Waldapfel opens the parapharyngeal space and cleans out the abscess, at the same time protecting the mediastinum. Kissling obtains good results by simply opening the parapharyngeal space and draining the abscess. He ligates the jugular vein above and below the facial vein and opens the latter. In some cases the thrombus may extend to the jugular bulb or even upward, so that it may become necessary to open the mastoid process to remove the sinus plate and expose the lateral sinus. Meningitis may be caused by involvement of the jugular bulb and the lateral sinus in this condition. In every case of sepsis following angina, the parapharyngeal space should be explored. There is a difference of opinion as to whether the tonsils should be removed at the time of operation.

After reviewing the literature, particularly the work of Goerke, Linck and Waldapfel, Hybášek [42] describes in detail the modus operandi of a thrombophlebitis secondary to tonsillectomy, also reviewing particularly the surgical treatment about the neck for this serious condition. The real interest in the article is the case report. So complete are the history and the record of the course, as well as the data on the different examinations of the blood, that it reads more like a novel than a case report. The best part of the report is the complete recovery of the patient, following a type of pyemia that has been observed by the majority of workers to be fatal. A striking thing in the report of the case is the fact that operation was performed on the tonsils only three weeks after severe acute lacunar tonsillitis of a streptococcic nature.

A survey of the literature on septicopyemia following tonsillar and pharyngeal infections was presented by Abt,[43] with a clinical discussion of the atria of infection from the throat, and the pathology, etiology, clinical course, complications, prognosis and treatment of the condition. Four cases are reported briefly, and the frequency of thrombosis of the internal jugular vein is emphasized. The treatment recommended is mostly surgical, drainage of the septic focus and ligation of the jugular vein on the affected side if there are chills and irregular fever.

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42. Hybášek, J.: Pyemia Following Tonsillectomy, Otolaryng. slavica **4**:137, 1932.

43. Abt, Isaac A.: Postangial Sepsis, J. Pediat. **1**:8, 1932.

Albrecht [44] reports 2 cases of posttonsillitic septicemia. In 1 case a septic thrombosis of the jugular vein developed which extended centrally to the superior thyroid vein and peripherally to the torcular herophili. The patient's life was saved after extensive opening of the vein, although metastases had developed in the pleura. The patient in the other case died of severe sepsis and numerous metastases in the lung, although several hours after the first chill the facial vein was ligated, the jugular vein was excluded by excision of a large piece and the parapharyngeal space and peritonsillar tissue were opened. No thrombi were found in the veins; histologically there were signs of acute endophlebitis. Thrombus formation is considered a favorable reaction of the venous walls; its absence resulted in death in 1 of the cases. This may have been due to the greater virulence of the organism, but it may also have been due to a weaker capacity of the venous wall for protective reaction. The author thinks that in asthenic persons the sinus walls are frequently thin and have little resistance to infection. This may also be true of the venous walls and may be of equal importance in the development of sinus phlebitis.

Greifenstein [45] presents a study of 4 cases of tonsillar and peritonsillar infection that led to thrombophlebitis of the large veins of the neck and fatal sepsis. He compares with these a case of thrombophlebitis and sepsis following otitis media, a case following the extraction of a tooth, and 2 cases following nasal furuncle. The tonsillar infection led to diffuse phlegmonous lymphangitis of the deep tissues of the neck, and this in turn to phlebitis and thrombosis of the large cervical veins, from which the infecting organisms entered the blood stream. Venous involvement may lead to discrete intimal inflammatory lesions over which multiple discontinuous thrombi form. In the cases of sepsis following otitis media and extraction of a tooth, spread of the infection to the lungs was ascribed to propagation of the thrombophlebitic process into the collaterals of the jugular vein. In the 2 cases of nasal furuncle, the process was propagated along the vena angularis and the vena ophthalmica to the cavernous sinus.

In Wessely's [46] opinion, endocranial complications after tonsillitis and peritonsillitis develop from direct spread of the tonsillar infection outward through the pharyngeal wall and through the parapharyngeal tissue up toward the base of the skull and by way of the oval foramen into the middle cranial fossa. Surgical therapy, if one wishes to prevent

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44. Albrecht, W.: Thrombophlebitic Septicemia, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:258, 1933.

45. Greifenstein, A.: Cervical Thrombophlebitis and Sepsis Secondary to Tonsillar Infection, Virchows Arch. f. path. Anat. **281**:748, 1931.

46. Wessely, E.: Endocranial Complications Following Tonsillitis and Peritonsillitis, Monatschr. f. Ohrenh. **66**:1190, 1932.

threatened endocranial complications, must consist in stopping the infectious process while it is still in the loose connective tissue of the parapharyngeal space. The recognition of inflammatory changes in the parapharyngeal space is therefore of paramount importance; among the symptoms are peritonsillar abscesses under tension and changes in the lateral pharyngeal wall and also on the outside of the neck. With the upward spread of the inflammatory process in the pharyngeal connective tissue spaces, symptoms from the adjacent muscles and nerves usually develop. Rigidity of the jaw is usually found. This may also be found in peritonsillar abscesses. If the rigidity of the jaw disappears while the local symptoms of a peritonsillar abscess continue, it usually indicates perforation of the abscess into the parapharyngeal space. Some of the subjective symptoms are irritation of the trigeminal and lingual nerves. In the author's opinion, the danger of endocranial complications justifies surgical intervention in cases of probable infection of the parapharyngeal space. Incision of the peritonsillar abscess and possibly tonsillectomy are valuable, but must be supplemented by opening of the parapharyngeal space. This may rarely be done from the interior; usually it is accomplished from the exterior. The author describes the method used at his clinic for the opening of the anterior paraphyngeal space.

Ascending infection of the endocranium with tonsillogenous pharyngeal phlegmon was observed by Vogel [47] in 3 cases which he reports. In 2 of the cases, following tonsillitis, a peritonsillar abscess developed, accompanied by septicemic symptoms, a pronounced painful swelling of the temporal region and meningitis which terminated fatally. In the third case the disease started with osteomyelitis of the right superior maxilla, the hard palate and finally the nasal septum. This was followed by swelling of the left side of the soft palate with thickening of the anterior and posterior palatine arch. The left temporal region was swollen and symptoms of meningitis and paralysis of the abducens nerve appeared. The patient recovered from these symptoms in three weeks but died later of an abscess of the left frontal lobe. A simple phlegmonous spread of the infection from the peritonsillar region is contraindicated by the absence of purulent infiltration in the parapharyngeal space. In all 3 cases thrombosis of part of the cervical veins was found. In the first case it occurred in the branches of the right anterior and posterior facial veins and the veins of the cavernous and intercavernous sinuses. In the second case there was thrombophlebitis of the left jugular vein and the veins of the left pterygopalatine fossa up to the cavernous sinus, the left jugular bulb and the sigmoid sinus. In the third case, thrombophlebitic changes were found in the partially excised facial

47. Vogel, K.: Ascending Infection of the Endocranium with Tonsillogenous Pharyngeal Phlegmon, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:4, 1933.

vein. The author calls attention to the characteristic painful swelling in the temporal region which probably results from infection of the pterygoid plexus and the venae temporales profundae communicating with it. This ominous swelling is an early manifestation of the ascending path of the thrombophlebitic infection, which cannot be stopped by ligation and probably always terminates fatally, as in the cases reported.

Elsaesser [48] says that the typical circumscript peritonsillar phlegmon which does not spread beyond the lateral pharyngeal wall rarely leads to serious complications and has a benign character in spite of its disturbing manifestations and in spite of the conception that most cases of posttonsillitic pyemia are preceded by a spreading of the virus through the peritonsillar tissue. In 160 cases of peritonsillitis observed at the author's clinic there was no serious or septic pyemic complication. The author thinks that the fact that the spread of tonsillar infection usually is by way of the submucosa is favorable for prognosis, as the strong inflammatory reaction produced in this tissue limits the zone of infection and prevents its extension. Why this reaction is sometimes absent and severe sepsis results is not known; the cases reported occurred mainly in healthy, resistant persons. The author found that the more intense the tissue reaction was, the earlier the abscess could be diagnosed, with the result that intervention could be undertaken in time to prevent the suppuration from extending into the parapharyngeal space. The danger of posttonsillitic septicemia is greatest if the abscess shows only slight extension, perhaps having a deep retrotonsillar position, and a reaction of the peritonsillar tissue is entirely or partly lacking, or when the infection does not spread to the peritonsillar tissue but invades the blood stream directly. Under these conditions, the cause of the septic manifestations remains undiscovered until too late for a curative intervention. The majority of cases of posttonsillitic septicemia reported in the literature did not exhibit peritonsillitic symptoms. The author thinks that many cases described in the literature as instances of complications after peritonsillitis may have involved lateropharyngeal abscesses which developed following tonsillitis without intermediary involvement of the peritonsillar region.

Linck [49] again affirms that the treatment of a peritonsillar abscess should consist in immediate tonsillectomy instead of incision of the abscess. Only by removal of the primary seat of infection, the tonsil, can the recurrence of peritonsillar abscesses be prevented. The substitution of tonsillectomy for incision of the abscess not only avoids a second prophylactic intervention but constitutes the best surgical therapy

48. Elsaesser, R.: Peritonsillitis, Arch. f. Ohren-, Nasen- u Kehlkopfh. **133**: 61, 1932.

49. Linck, A.: Peritonsillar Abscess and Tonsillectomy for Abscess (Winkeler's Operation), Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:227, 1933.

for the abscess. Resection of the tonsil, which usually forms the front wall of the abscess, effectively drains the abscess whether it is a small concealed one or a large ripe one. No spreading with the dressing forceps and no tamponade are necessary. Only in extremely rare cases in which the abscess has developed in the muscular layer are trial puncture and incision necessary. The exposure of the posterior wall of the abscess by tonsillectomy insures rapid healing and guards against the spread of the suppuration to the cervical veins. The danger of propagating the infection by tonsillectomy during acute inflammation of the tonsillar region has been disproved by experience. The opening of lymph channels and tissue spaces occurs just as much in incision of the abscess. More than 200 tonsillectomies for abscess in the author's clinic have not resulted in a single complication due to the intervention. The danger of spreading the infection, attributed to local anesthesia, is avoided by a combination of regional superficial anesthesia from the exterior and superficial infiltration anesthesia from the interior. Hemorrhages are less frequent in tonsillectomy than in incision and are more easily stopped by acupression than by the tamponade used in hemorrhage from incisions.

Two cases are reported by Wilson and Darkes [50] in which they consider optic neuritis to have been due clearly to infection of the tonsils.

The removal of the tonsils, in both cases badly diseased, produced prompt improvement and a return in a relatively short time to normal vision and a normal state of the involved nerve.

If swelling of one or both optic nerves is the only physical sign present, and if the patient does not have the headache or other symptoms suggestive of a tumor of the brain, he should not be rushed into submitting to encephalography, ventriculography or a decompression operation.

Siegmann [51] discusses the literature and reports 12 cases of inflammatory complications of the wound following tonsillectomy that occurred in Hajeck's Clinic between 1926 and 1931 and a few others. The series comprises 5 cases of inflammatory infiltration of the wound; 1 case of pneumonia with inflammation of the wound, followed by otitis; 4 cases of peritonsillar abscess; 4 cases of parapharyngeal or retropharyngeal abscess, and 1 case of infiltration toward the mediastinum. The author thinks that these postoperative inflammatory complications can probably be attributed to faulty anesthetizing technic, consisting in an injection into diseased tissue or the carrying of bacteria from the surface of the mucosa into the submucous tissue with the hypodermic needle. By

50. Wilson, G., and Darkes, W. F.: Unilateral Choked Disk Due to Chronic Tonsillar Infection, J. A. M. A. **100**:1233 (April 22) 1933.

51. Siegmann, N.: Inflammatory Complications of the Wound Following Tonsillectomy, Monatschr. f. Ohrenh. **66**:1458, 1932.

injecting too deeply one may cause local infection either of the pharyngeal musculature or of the parapharyngeal space (by piercing the pharyngeal wall), with danger of spread in several directions, particularly toward the mediastinum. The author emphasizes the importance of waiting four weeks after the last attack of tonsillitis.

Hinsberg [52] reports a personal observation of an acute infection of the "tonsillar region" accompanied by nephritis several years after tonsillectomy and 4 cases of peritonsillar abscesses occurring despite tonsillectomy, 2 of which are personal observations while 2 are cases from the literature. These 4 cases show that despite tonsillectomy, and sometimes several years later, an abscess may be formed in the region of the scar which presents the typical picture of a peritonsillar abscess and which may heal spontaneously or after incision or may lead to severe sepsis and death. In 1 of the 2 cases from the literature a small part of the superior tonsillar pole remained after operation; in the other case also this probably occurred. In 1 of the author's cases a piece of tonsillar tissue half the size of a pea was found in the scar; in the other case such a fragment appeared to have remained at the lower pole. These tonsillar remnants are probably to be considered as the focus for the abscess formation. How infection occurred is not certain. The practical interest of these observations lies in the fact that abscess formation may occur even after tonsillectomy and in the warning contained in them to avoid leaving even the smallest tonsillar remnants when performing tonsillectomy, especially on the upper pole.

Wirth and Renno [53] report that of the 2,766 patients on whom tonsillectomies were performed in their clinic from 1920 to 1932, the majority remained free from complications. Hemorrhages developed in 2.3 per cent of the cases, increases of temperature to over 37.5 C. (99.5 F.) were observed in 5.8 per cent, and bronchopneumonia developed in 0.2 per cent. From this the authors conclude that, although tonsillectomy is not a "dangerous" operation, it is not entirely harmless, for there were 2 fatalities, resulting from hemorrhage and from hemorrhage and bronchopneumonia, respectively. Before deciding in favor of a tonsillectomy, the general condition and age of the patient, the danger of hemorrhage and whether the expected advantages warrant the exposure of the patient to possible dangers should be taken into consideration.

Brauer [54] gives a comprehensive discussion of hemorrhages following tonsillar disease, based on the literature and on 9 personal observa-

52. Hinsberg, V.: Tonsillitis with Nephritis and Peritonsillar Abscess Despite Tonsillectomy, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:77, 1933.

53. Wirth, E., and Renno, G.: Dangers of Tonsillectomy, Deutsche med. Wchnschr. **59**:322, 1933.

54. Brauer, W.: Hemorrhages Following Tonsillar Disease, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:558, 1933.

tions. Such hemorrhages occur chiefly in inflammations in the peritonsillar tissue. It is often difficult to determine which vessel has been eroded. In 33 of the 72 cases in the literature, the hemorrhages were healed—in 7 cases spontaneously or by tamponade, in 3 by tonsillectomy, in 10 by ligature of the external carotid artery and in 13 by ligature of the common carotid. Of the 39 patients who died, 30 received no treatment. While these figures show the value of ligation in severe cases, the author does not think that it is indicated in every case of hemorrhage; but he insists on hospital supervision, so that no time will be lost if surgical intervention becomes necessary. Early incision of abscesses is a prophylactic measure. General rules for therapy are discussed. In slight hemorrhages tamponade may be employed. If the exact location is known, acupression, sometimes after tonsillectomy, may be used; in other cases tonsillectomy alone stops bleeding. Such hemorrhages usually come from branches of the external carotid artery. Severe hemorrhages demand ligation of the vessel concerned. If compression of the external carotid stops the hemorrhage, only this artery need be ligated. If compression does not stop the bleeding or decrease it materially, the internal carotid is involved; and the common carotid must be ligated, as the branches of the external carotid also usually terminate in the abscess, and ligation of the internal carotid alone would not suffice. If ligation is performed after the hemorrhage has stopped, the external carotid is ligated and a loose ligature applied to the common carotid (its end issuing from the wound), which may be constricted if necessary. After ligation of the vessels, the phlegmon in the pharynx must be incised. In rare cases in which ligature of the common carotid is not effective because of hemorrhage from the peripheral vascular region or anastomoses of the other side, tight tamponade must be used. If there is the slightest cause for suspecting an aneurysm, a loose ligature must be placed around the common carotid before the phlegmon is incised so that a fatal hemorrhage may be prevented. For small venous hemorrhages, the same therapy as for small arterial hemorrhages is indicated. Tight tamponade will often stop hemorrhages even from the internal jugular vein. Hemorrhage from ulcerations in the tonsils and hemorrhage from the tonsils due to general disease are briefly discussed.

According to Babbitt,[55] hemorrhage, anesthetic shock and pulmonary involvement are the three grave complications which may follow the operation for the removal of tonsils and adenoids. He says that it would be natural to assume that pulmonary complications occur through

55. Babbitt, James A.: Pulmonary Complications of Tonsillectomy with Report of Three Cases, Arch. Otolaryng. **17**:118 (Jan.) 1933.

some carelessness in routine or some predisposing pathologic process. Yet after thirty years of his active service in connection with operations on the tonsils in several hospitals, with scarcely a case of definite pulmonary complication as a sequence, 3 cases of such complications, 2 of lung abscesses and 1 of pneumonia, occurred in the two and one-half months prior to his report in the service under his direction; and these complications followed what were reported as perfectly normal tonsillectomies and adenoidectomies. Operations were done after routine reference from the outpatient department, with the customary preliminary safeguards performed in each case by a different operator, under ether anesthesia, with the patient in the prone position and with the use of the suction tube.

An interesting and instructive article on a study of 24 cases of infection of the neck was published by Beck.[56] An analysis of the etiology of these cases showed that acute or subacute inflammation of the tonsils preceded or accompanied the attack in 11 cases. In several cases the tonsillitis occurred from one to two months prior to the outbreak in the neck. Inflammation of the pharynx alone accounted for 4 cases. In these, the tonsils had been removed from one to three years previously. It was noted that the infection could enter through the pharyngeal wall. Pharyngeal and tonsillar inflammations accounted for 14 of the cases. Other cases followed infections in the teeth, and still others followed operations on the tonsils and adenoids. An investigation of the bacteriology of these cases revealed that the hemolytic streptococcus was responsible for about 50 per cent. The author discusses the applied anatomy and regional manifestations, for example, the infection as located in the pharyngomaxillary space, the submaxillary space and the carotid sheath, cervical lymph node suppuration, prevertebral fascial infection and buccopharyngeal, pretracheal and visceral fascial infection. He then outlines the various surgical operations for drainage. Finally he discusses the complications, such as rupture of the abscess through the external auditory canal, thrombosis of the jugular vein and numerous other manifestations. It is noteworthy that phlebitis of the jugular vein occurred in 20 per cent of the cases. It is emphasized that these cases of jugular thrombosis from infection in the planes of the cervical fascia are going unrecognized and patients are dying from septicemia. The absence of frank local manifestations of the presence of infection, notably swelling, is common. Delayed surgical intervention is apt to be disastrous and engenders regret.

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56. Beck, A. L.: A Study of Twenty-Four Cases of Neck Infection, Ann. Otol., Rhin. & Laryng. **42**:742, 1933.

Voss [57] reports the case of a man, aged 23, in whom tonsillectomy was followed by suppuration of a wisdom tooth, succeeded by phlegmonous inflammation of the soft parts of the cheeks and throat and finally osteomyelitis of the inferior maxilla. The author thinks that if there was any relation between the tonsillectomy and the succeeding manifestations, it could have been only that the tonsillectomy had a sensitizing influence on the latent infection of the wisdom tooth. It is equally probable that it was merely an unfortunate accidental succession of the two processes. In any event, this case shows again the importance of scrupulous examination of the teeth and especially of a cutting wisdom tooth before tonsillectomy, so that all pathologic anomalies may be removed before the intervention.

Bernfeld [58] says that rigidity of the neck after adenotomy, recently reported by Schaeffer as a rare complication, is not so rare as the latter supposes. He reports 2 cases. In both of them, as in Schaeffer's, there was no significant demonstrable glandular swelling. As malformation of the epipharynx existed in both cases, Bernfeld thinks that an injury of the deeper muscles incurred during intervention in the constricted nasopharynx resulted in myositis and consecutive rigidity of the neck.

TREATMENT

Smith [59] brings up the question whether fusospirochetal infections should be treated with arsenicals. He emphasizes that arsenicals administered intravenously are not harmless; therefore he believes that local treatment should be tried first. Intravenous treatment, however, with arsenicals is indicated for patients who do not respond to local treatment and for those whose lungs have become involved. Small doses of from 0.2 to 0.3 Gm. repeated at intervals of from two to four days are less dangerous and quite as effective as the larger doses. Finally he concludes that "there is clinical evidence for the belief that the fusospirochetal mixture of anaerobic organisms is pathogenic." If one can consider a group of anaerobic organisms acting in symbiosis as a single entity, Koch's postulates have been fulfilled: (*a*) The fusospirochetal organisms are constantly present in the lesion; (*b*) they have been isolated in pure culture; (*c*) when recombined and injected into animals, they produced the typical disease; (*d*) they have been isolated from the experimental lesions; (*e*) up to the present, the

57. Voss, O.: Osteomyelitis of Inferior Maxilla Following Tonsillectomy and Consecutive Extraction of Wisdom Tooth, Ztschr. f. Laryng., Rhin., Otol. **22**:474, 1932.

58. Bernfeld, K.: Rigidity of Neck Following Adenotomy, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:293, 1932.

59. Smith, David T.: Should Fusospirochetal Infections be Treated with Arsenicals? Arch. Otolaryng. **18**:760 (Dec.) 1933.

typical disease has not been produced by any other organism or any other combination of organisms. The use of intravenous injections of neoarsphenamine or sulpharsphenamine is the most effective treatment for the more severe and the chronic infections with fusospirochetal organisms.

It is McHenry's [60] custom to consider a diagnosis of Vincent's infection of the tonsil confirmed when the lesions respond within twenty-four hours to treatment with copper sulphate and sodium perborate. When they do not respond, he resorts to smears, cultures and dark-field examinations. If the history and clinical examination suggest diphtheria or syphilis or an acute pyogenic or streptococcic infection, he uses the microscope and the culture tube. To justify this procedure the author studied his last 40 cases of Vincent's tonsillitis in regard to history, clinical appearance and response to treatment. The average amount of time required for the relief of symptoms was two days. Eleven patients were relieved from symptoms in one day, and 1 required four days before local relief was obtained. The average amount of time required for the disappearance of local swelling was two and four-tenths days. Complete disappearance of the membrane was achieved in the average time of four and six-tenths days. Four patients required only two treatments each; 4 had seven treatments each, and 1 had ten treatments. There were nine recorded recurrences in 6 patients. In employing this procedure in Vincent's tonsillitis the author removes the necrotic débris and the membrane, which separates easily, by a mild alkaline spray and gentle wiping with a cotton-tipped applicator. He applies a 10 per cent aqueous solution of copper sulphate to all ulcerated surfaces. This should reach the depths of all pockets and infected crypts. This procedure is repeated every day until the signs of acute inflammation have disappeared, and every second day until the membrane has disappeared completely. The patient is given a prescription for sodium perborate and instructed to use one-half teaspoonful in half a glass of warm water as a gargle and mouth wash every two hours.

After reviewing some of the methods formerly recommended for the disinfection of diphtheria carriers and after pointing out some of their shortcomings, Meyer [61] describes his own method. He applies a 3 per cent alcoholic solution of methyl violet by means of a cotton compress to the tonsils and to the nasal mucous membrane. This solution not only spreads rapidly over the surface but enters the folds and crypts. The applications are made from two to three times weekly,

60. McHenry, L. C.: Treatment of Vincent's Tonsillitis with Copper Sulphate and Sodium Perborate, South. M. J. **26**:193, 1933.

61. Meyer, A.: Disinfection of Diphtheria Bacillus Carriers by Means of Methyl Violet, J. A. M. A. **28**:1563, 1932.

and sterilization is obtained generally after seven or eight days and at the latest after seven weeks. The disinfection is considered complete after three smears (taken at intervals of eight days) have proved to be free from bacilli. The procedure was effective in 19 of 20 carriers. In spite of this comparatively small amount of material, the author considers his method worthy of further trial, because of the high percentage of efficiency and because it is simple, does not involve dangers and can be carried out without great expense.

Layton [62] does not believe that there is as yet any local treatment of the tonsils that can compare with operative intervention. Size is no indication for removing tonsils. The great indication is the history of recurring follicular tonsillitis, and this is more important than the appearance of the tonsil. Children need lymphoid tissue in their respiratory tracts, and they do not get follicular tonsillitis in the earlier years of life. Therefore, operations on a child under 5, 6, 7 or 8 years of age should not be done unless it cannot possibly be helped and unless the need for it can be proved. After that age the operative treatment of the tonsils is a practical problem, but the author thinks that the best results come after the age of 12 rather than before.

Cemach [63] thinks that in many cases of the adenoid syndrome the same therapeutic result can be accomplished by adenotomy alone as by the customary combined adenotomy and tonsillectomy. He reports that of 154 children on whom he performed adenotomy alone, although the tonsils exhibited more or less alteration, 68 per cent were completely cured of symptoms and 10 per cent were clinically improved to the extent that the parents did not wish further therapy; in only 21 per cent the symptoms were not influenced by adenotomy and tonsillectomy had to be performed. Correlating these results with the classification of the cases according to tonsillar symptoms, the author concludes that simple hypertrophy (cases of excessive size excepted) is not an indication for tonsillectomy. In recurring acute pharyngotonsillitis only adenotomy should be performed at first. In chronic tonsillitis also it is permissible to essay adenotomy first, provided there are no general symptoms, such as fever or sign of involvement of the heart, joints or kidneys.

Neumann [64] says that adenoid growths in infants who feed at the breast or on a bottle may obstruct nasal breathing to such a degree

62. Layton, T. B.: Treatment of Enlarged Tonsils in Children, Lancet **1**:509, 1933.

63. Cemach, A. I.: Indications for Tonsillectomy in Childhood, Monatschr. f. Ohrenh. **66**:1453, 1932.

64. Neumann, H.: Adenoids and Adenotomy in First Weeks and Months of Life, Monatschr. f. Ohrenh. **66**:922, 1932.

that sucking is interfered with and nutrition is disturbed. In the majority of these cases medical treatment will permit postponing adenotomy until the end of the first half year or year, at least beyond the first weeks or months, of life; but in some cases surgical intervention becomes an immediately necessity. The author thinks that adenotomy is indicated if a palpable tumor filling the nasopharynx has been ascertained, if interference with feeding and nutrition results from the obstruction of nasal breathing, or if there is retarded development attributed to adenoids by the attending pediatrician. Three distinct types of adenoids occur in nurslings. The first is simple hyperplasia without accumulation of secretion. The second is irregular hypertrophy showing the beginning of the villous form frequently seen in older persons. This form usually has a heavy tenacious mucoid secretion and is often accompanied by nasal hypersecretion. The third form has a relatively small tumor, but heavy secretion in the nose and nasopharynx. Diagnosis in infants only a few weeks or months old is difficult. The author employs palpation with a sound introduced by mouth and indirect palpation with the finger to determine the resistance when the uvula is pressed back. The pressing down of the rear part of the tongue with a small laryngeal mirror frequently permits seeing the adenoid growths in the initial moment of the vomiting reflex. The author recommends instillation of oil for one or two weeks before adenotomy whenever possible. He describes the technic of performing the operation with his modification of Beckman's curet. This curet possesses a prong which is pushed into the tumor, holding it firm. It is essential to use a sharp instrument and perform the operation in one stroke. An anesthetic is not used for children under 1 year of age. Hemorrhage is slight. The results of this intervention in nurslings are usually excellent.

Summarizing his results with the so-called "tonsil and adenoid operation," Love [65] concludes:

1. Adenoids should be removed early, and the best results are attained in the cure of mouth-breathing if the operation is followed by training of the patient to overcome the acquired habit.

2. Tonsils may often be left unless they are actually septic.

3. A discharge from the middle ear due to other causes, for example, scarlet fever and measles, is not removed by the operation on the tonsils or the postnasal adenoids.

4. Nasal discharge is not always cured by such operations, but even in cases of apparent failure great improvement takes place.

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65. Love, James Kerr: Results of the So-Called Tonsil and Adenoid Operation, Lancet **1**:1356, 1932.

From the results of 736 tonsillectomies and the observations on 741 children who refused operations, Monroe and Volk [66] conclude and summarize their findings as follows: 1. Greatest relief is obtained from sore throats, head colds, mouth-breathing and malnutrition. 2. Complaints were relieved in the first six months in 91 per cent of the cases. 3. Operation in early childhood offers the best results. 4. Forty per cent of the mentally subnormal or retarded children were improved. 5. Coexisting conditions, except probably pulmonary tuberculosis, are not contraindications for tonsillectomy. 6. The control group showed no definite improvement in a similar period of observations. Excellent tables are furnished.

From a review of the literature, Tschiassny [67] concludes that the therapeutic value of irradiation of the tonsils and its dangers are not sufficiently well established to form a basis for indications for this therapy. The author thinks that it is not sufficiently innocuous for a conservative form of treatment and that, as a treatment that is not innocuous, it is not rapid and reliable enough to be generally accepted. It is indicated chiefly in cases in which there are strong contraindications to surgical intervention. The author studied the morphologic effect of roentgen irradiation on the tonsils in a case in which one of the tonsils was removed by tonsillectomy at one sitting and the other was enucleated five weeks later after three roentgen irradiations. The patient was a child, aged 5 years; both tonsils before intervention exhibited hypertrophy of medium degree. On removal, the irradiated tonsil was found to be much larger than the other; the increase in size affected chiefly the segment hidden in the tonsillar fossa. The microscopic picture was characterized by a conspicuous enlargement of the germinal centers, which appeared loosened and heterogeneous in structure and exhibited much proliferation, giving the appearance of a neoplastic growth. This observation contradicts the experience of roentgenologists. Longer irradiation might have given a different result.

Zoepffel [68] reports his results with roentgen treatment of 50 children with hypertrophied tonsils. Good results were obtained in 68 per cent of the 38 children with large, soft tonsils and in 25 per cent of the 12 children with firm, fibrotic tonsils. A result was considered good if the tonsils became smaller and there was an improvement in the subjective symptoms, such as anorexia, impairment of hearing, difficulty in breath-

66. Monroe, J. D., and Volk, V. L.: A Study of the Effect of Seven Hundred and Thirty-Six Tonsillectomies and Seven Hundred and Forty-One Controls, Rep. Am. Child Health A. **6**:15, 1929.

67. Tschiassny, K.: Roentgen Irradiation of Tonsils, Monatschr. f. Ohrenh. **66**:1444, 1932.

68. Zoepffel, H.: The Roentgen Treatment of Tonsils in Children, München. med. Wchnschr. **79**:1753, 1932.

ing, a tendency toward tonsillitis and pains in the joints. The treatments were repeated two or three times at intervals of five or six weeks. A few children with fibrotic tonsils were given five exposures. No ill effects were observed. There was no excessive bleeding among those who subsequently required adenoidectomy.

The treatment of malignant disease of the tonsil with high voltage roentgen rays and radium is reported by McKinney,[69] and he makes the following comments on this method of therapy:

> I am of the opinion that malignant disease of the tonsil is treated most satisfactorily by blocking with high voltage roentgen treatment, implantation of radon gold seeds, dissection of the neck in a thorough manner and further high voltage roentgen treatment in the order given. A radical attack on the glands, either by interstitial irradiation or by surgical removal, should never be made until the primary lesion is treated, for that lesion is the distributing point not only for the cancer cells, but also for possible infectious conditions.
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> It would be very dangerous if patients were subjected to high voltage roentgen treatment alone at the hands of indiscriminate roentgenologists, for this treatment is capable of good results only when applied in carefully regulated doses by a skilled roentgenologist. However, if Coutard's expectations are realized surgical intervention in these cases may eventually be supplanted by intensive high voltage roentgen treatment.
>
> My experience has been that of a rhinolaryngologist to whom these patients have come on account of conditions of the throat. I have felt happy to be so situated that I could have the aid of a competent general surgeon in this work and of the x-ray laboratory of the Baptist Memorial Hospital, which is well equipped for obtaining good results in the application of roentgen therapy.

According to Balmer,[70] the control of tonsillar bleeding in accordance with the basic principles of general surgery is essential in the removal of tonsils. Faulty operative position and lack of a definite precise technic are greatly responsible for the majority of unsatisfactory postoperative results of tonsillectomy. The author's technic is as follows: The tonsil is swabbed with a 1:1,000 epinephrine solution. The surface of the tonsil and the interior of the crypts are swabbed with a small amount of cocaine hydrochloride flakes by means of a fine applicator tipped with cotton and moistened with a 1:6,000 epinephrine solution, the excess of moisture being squeezed out. This is repeated two or three times at two or three minute intervals. The electrocoagulation apparatus is employed, the meter reading about 3,000 milliamperes with the spark gaps slightly open. This will give a reading of from 250 to 300 milliamperes with the patient in the circuit. The indifferent electrode is connected to the metal chair on which the patient is seated. The proper needle is inserted into the tonsil substance approximately

69. McKinney, Richmond: Treatment of Malignant Disease of the Tonsil with High Voltage X-Rays and Radium, Arch. Otolaryng. **18**:40 (July) 1933.

70. Balmer, F. B.: Electrocoagulation of the Tonsils: Is It a Commendable and Worthwhile Procedure? Northwest Med. **31**:470, 1932.

4 mm.; it should be kept about 4 or 5 mm. away from the peripheral structures; sparking and surface fulguration should be avoided, and the point should be directed toward the center of the fossa. In from one to three seconds a blanched area will appear around the needle. This process is repeated as many times as is necessary, usually from six to ten contacts being made. A small area of unblanched tissue which is left between the punctures allows the coagulated areas to coalesce and prevents overcoagulation and the possibility of too early separation of the coagulum. Bleeding points may be controlled by sparking the area. Electrosurgery, electrocoagulation in particular, does not replace surgery in the removal of tonsils. It is better suited to selected cases. The combination of surgery and electrosurgery is the ideal method. Ordinary surgery will continue to be the method of choice; when it is contraindicated, electrosurgery may be considered as an appropriate and scientific aid. The surgeon should not be limited by lack of ability, knowledge or equipment or by prejudice. There are many contraindications to electrocoagulation. Diathermocryptectomy, or removal of the cryptic portion of the tonsil, is a commendable, worth while procedure to be considered under circumstances in which a more conservative procedure is required. Electrocoagulation is a safe, ultraconservative procedure, requiring time, judgment, technical skill, patience and meticulous care.

Barlow [71] believes that electrocoagulation is an essential part of physical therapy. Electrocoagulation tonsillectomy, in his opinion, requires more care, judgment and dexterity than the approved methods of surgery. Many men of national repute have tried the method and after a week or two have discarded it and become confirmed antagonists. The stumbling blocks and pitfalls in electrocoagulation are: the apparent simplicity of the method; the sales talk of some salesmen, encouraging any one to buy a machine and use the method; the necessity of developing a technic on private patients without competent instruction; the tendency to hurry and coagulate too large an area at each treatment; the possibility of coagulating into the muscle and causing severe pain after treatment; the use of too much local anesthetic, and the possibility of causing a flare-up of old quinsy by not allowing sufficient time to elapse after an attack of tonsillitis.

Skillern [72] considers electrocoagulation a method of removing the tonsils that every laryngologist should master, as he will find it is necessary occasionally to remove them by this procedure. It should be spoken of not as the operation of choice but as the operation of

71. Barlow, R. A.: Pitfalls in Tonsil Coagulation, Arch. Phys. Therapy **13**: 598, 1932.

72. Skillern, S. R.: Is Electrocoagulation the Best Method for Removal of Faucial Tonsils? Arch. Phys. Therapy **13**:587, 1932.

necessity. It is the most difficult and dangerous method in the hands of the inexperienced. The dangers and difficulties are multiple in ratio to the lack of knowledge, and no one who is not capable of removing tonsils by other surgical technics and of meeting all complications that may arise during or after the operation should attempt their removal by electrocoagulation. For the average person, electrocoagulation with its multiple applications of the needle cannot be mentioned in the same category with that of a clean surgical tonsillectomy by a trained operator.

Halle [73] thinks that electrocoagulation of the tonsils, with its unavoidable discomfort for the patient and its injury to adjacent tissue, should be substituted for tonsillectomy only in cases of hemophilia in which the utmost precaution against dangerous hemorrhages is imperative. In all other cases electrocoagulation is contraindicated, especially in view of the fact that the dangers of hemorrhage can be almost eliminated by correct surgical procedure. He has designed two forceps which permit a firm grasp of the tonsil even if it is small and embedded. With these the tonsil may be pulled forward so that not much of the glossopalatine arch need be sacrificed in the excision of the tonsil. The technic is briefly described. If one strictly avoids using the instruments for holding the tonsil for handling other tissues or vessels, infection need not be feared, especially if an antiseptic powder is sprayed in the tonsillar fossa after operation.

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73. Halle: Tonsillectomy or Electrocoagulation of the Tonsils? Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:364, 1933.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Cleveland on June 11, 1934, during the annual meeting of the American Medical Association. Sixty-two candidates were examined, fourteen of whom were conditioned or failed.

The Board will hold an examination in Chicago on September 8, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology, and one in San Antonio, Texas, on November 16, during the meeting of the Southern Medical Society. Applications for certificate may be obtained from the secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Neb.

Abstracts from Current Literature

Ear

Cod Liver Oil Concentrate. W. D. Sutleff, E. H. Place and S. H. Segove, J. A. M. A. **100**:725 (March 11) 1933.

The use of cod liver oil concentrate (vitamins A and D) in daily doses of 100 minims (5.9 cc.) apparently had no effect on the incidence of otitis media in patients with scarlet fever. Bonar, Salt Lake City. [Am. J. Dis. Child.]

The Treatment of Acute Otitis Media in Acute Febrile Conditions. T. O. Graham, Brit. M. J. **2**:287 (Aug. 12) 1933.

In the early stage of acute otitis media abortive measures, such as rest in bed, administration of a purge, steam inhalations of tincture of benzoin, drops of warm glycerin and phenol solution and a rubber hot-water bag to the ear, may suffice to bring about resolution and cure. If this is ineffective, the drum membrane should be freely incised and adequate drainage of the tympanic cavity and of the mastoid antrum, via the aditus, afforded. This may bring about a restitution to normal of an infected mastoid. If fever persists in spite of adequate drainage and pain and tenderness in the mastoid increase, the patient is obviously not improving; mastoidotomy becomes imperative, and should not be unduly delayed. By adopting such a definite course one may hope to avert the onset of the more serious complications of otitis media, namely, meningitis, lateral sinus thrombosis, cerebral and cerebellar abscess, facial paralysis, labyrinthitis and deafness, the treatment of which is outside the scope of this paper.

Author's Summary. [Am. J. Dis. Child.]

Rôle of Mastoiditis in Toxicosis of Infancy. Ede v. György, Arch. f. Kinderh. **100**:238 (Oct. 31) 1933.

Twenty-seven cases in which antrotomy was performed in infancy are reported. In three cases there was uncomplicated mastoiditis, and in twenty-four cases, latent mastoiditis associated with symptoms of intoxication. The mortality in the latter group was 55 per cent. The infection of the antrum began in some cases at the onset of the intoxication, in other cases during the course of the intoxication and in still other cases during convalescence from the intoxication. In every case the antrum was found to be inflamed and edematous or to contain frank pus. Recovery after antrotomy was usually complete within from five to eight weeks. György believes that bilateral antrotomy is indicated when the cause of intoxication is not apparent, even though examination of the ear does not show definite signs of mastoiditis.

LeMaster, Evanston, Ill. [Am. J. Dis. Child.]

Rotation of the Eye in Normal Persons Measured on Fundus Oculi by Grahe's Method. P. H. Opfer, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:298 (Oct. 14) 1933.

Opfer measured the rotation of the right and of the left eye by Grahe's method in fifty complete examinations on twenty-one normal persons. The test subject, strapped to Grahe's table, was turned (slowly and without jerking) around the sagittal axis toward the side to angles of 20, 40, 60 and 90 degrees and back again. The rotation of the eyeball at each of these angles was measured. The test was done on both sides. Irregular variations of the rotation were observed in repeated tests in the same person, so that the values obtained in a single test

may not be generalized. In lateral inclination of a patient from the vertical position, the homolateral eye (right eye in inclination to the right) makes a stronger counterrotation than the heterolateral eye. This shows that in the case of positional stimulus as in the case of rotation stimulus the individual labyrinth acts with different intensity on each eye. The average maximum rotation occurs at an angle of 60 degrees. In inclination to the right the maximum rotation was usually produced at an angle of slightly less than 60 degrees whereas on inclination to the left it was usually reached at an angle of more than 60 degrees but never more than 90 degrees. In this respect it agrees with the rotary stimulus as the postrotatory nystagmus also exhibits different values for right and left rotation. The average maximal angle of torsion for the right eye is 5.3 degrees and for the left eye is 5.7 (maximum 2; minimum 11). In rotation from the horizontal position to the vertical the angle of torsion is slightly less than in rotation starting from the vertical position. In 66 per cent of the test subjects in turning from the horizontal position to the vertical position the eye returned to its original position. In half of the remainder of the persons the eyeball did not return all the way to the original (zero) position; in the other half the counterrotation of the eye went beyond the zero position.

EDITOR'S ABSTRACT.

DISTURBANCES OF LABYRINTHINE FUNCTION AFTER TRAUMA OF THE SKULL WITH AND WITHOUT INJURY OF THE TEMPORAL BONE. J. KOCH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:105 (Nov. 24) 1933.

For four years Koch examined periodically 146 patients who complained of disturbances in hearing and equilibrium following cranial trauma. The cases comprised 85 cranial fractures with demonstrable injury of the temporal bone, 14 cranial fractures without demonstrable injury of the bone and 47 cases of cranial trauma without fracture (cerebral and labyrinthine concussion). On the basis of this study Koch discusses at length the development of labyrinthine disturbances after cranial trauma and especially their evaluation and the question of the restoration of disturbed labyrinthine function. Unilateral total deafness existed in 30 cases, middle ear deafness in 33, labyrinthine deafness in 37 and combined deafness in 36. Middle ear deafness was found almost exclusively in the cases of direct injury of the middle ear or mastoid process. Labyrinthine and combined deafness were observed equally in all forms of trauma. Disturbances of vestibular function were found in 72 cases, 69 times in combination with cochlear disturbances and 3 times alone in the form of differences in tonus. The vestibular symptoms also occurred in all of the types of trauma. Cranial trauma may be followed immediately or later by differences in vestibular tonus. The author regards these as vasomotor disturbances in the central vestibular sections caused by the trauma but favored by previously existing vasoneurosis. The restoration of vestibular function varies in respect to time and manner of occurrence and may not occur at all. The constitution of the person is probably the determining factor. In a large number of cases function is not restored in two or three years, as is commonly assumed. If, in the presence of complaints about disturbances in equilibrium, significant changes of experimental excitability are observed, even though more than three years have elapsed since the trauma, disability due to disturbances in equilibrium must be conceded. The author's observations lead him to agree with Wittmaack that severe disturbances of labyrinthine function, including total deafness, do not depend on fracture of the skull but may result from cerebral concussions.

EDITOR'S ABSTRACT.

CLINICAL AND ANATOMIC EXAMINATION OF THE EAR IN A CASE OF INTERNAL HYDROCEPHALUS. H. BRUNNER, Monatschr. f. Ohrenh. **67**:1309 (Nov.) 1933.

Brunner reports the case of a girl, aged 10 years, who died of acute exacerbation of chronic internal hydrocephalus which was not diagnosed until necropsy because the symptoms in most respects resembled those of lethargic encephalitis.

The latter diagnosis was, however, contradicted by disturbances of the ocular muscles, the absence of rigidity, bilateral choked disks and the gradual loss of labyrinthine function. Microscopic examination disclosed that the focal symptoms were caused by degenerative changes of the motor nuclei of the brain stem. Fresh otitis media existed in both ears. The labyrinths exhibited no changes attributable to hydrocephalus, such as stasis in the blood vessels, extravasations into the perilymphatic or endolymphatic spaces, degeneration of the sensory cells or dislocation of Reissner's membrane, despite pronounced cerebral edema. The cochlear function appeared to remain normal till death; at least the rapidly progressive increase of intracranial pressure produced no noticeable defect of hearing. This pressure resulted at first in vestibular hyperexcitability, but this changed later to hypo-excitability (absence of the rapid component of nystagmus). This case shows that the effect of increased intracranial pressure on the optic nerve is opposite to the effect on the acoustic nerve. The most severe changes in the optic nerve were found in the peripheral portion, while the central paths were spared. The acoustic nerve exhibited no peripheral changes, but the central paths of the vestibular nerve were diseased, resulting at first in hyperexcitability, then in hypo-excitability, of the labyrinth.

EDITOR'S ABSTRACT.

THE TEMPORAL BONE AND ITS SURROUNDINGS. G. CLAUS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:515 (Oct. 14) 1933.

Claus examined the gross anatomic characteristics of 1,500 temporal bones. He discusses the results of his study with especial reference to otologic variations of practical importance to otologists.

EDITOR'S ABSTRACT.

DARWIN'S EAR AND ITS COSMETIC TREATMENT. E. EITNER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:564 (Oct. 14) 1933.

Eitner discusses the deformity of the ear known as Darwin's ear and its variations and the cosmetic treatment of each type.

EDITOR'S ABSTRACT.

NYSTAGMOGRAPHY DURING ROTATION. J. KUILMAN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:85 (Nov. 10) 1933.

Kuilman states that none of the present methods of nystagmography enable one to obtain curves during rotation from which one can read, in addition to the frequency, direction and rapidity, the form and evolution of the horizontal, vertical and rotatory nystagmic movements. He describes an apparatus with which this is possible. With this device not only horizontal, vertical and rotatory ocular movements, but also the compensatory positions of the eye (rotation) can be registered during rotation around different axes and in different positions. He reports the results of 140 curves obtained in tests on normal persons. Accelerated speed of rotation in rotations of about 90 degrees does not increase the frequency of the nystagmus, but the slow phase becomes more rapid. Analysis of the nystagmus curves obtained in rotation about the horizontal longitudinal body axis showed that the rotation of the eye affects the direction of the nystagmus. The rotation of the eye appears gradually during the occurrence of the nystagmus.

EDITOR'S ABSTRACT.

INVOLVEMENT OF THE EAR IN CAISSON DISEASE. JOHANNA KORTE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:349 (Nov.) 1933.

Korte states that the manifestations of caisson disease in the ear fall into two groups: (1) those of purely mechanical origin occurring either during compression or decompression and (2) those which frequently constitute only a part of a disease affecting the whole body and which are due to air embolism. Air embolism which results from decompression manifests itself a short time after the subject

has left the caisson in the symptoms of Ménière's disease, and apoplectiform deafness or baryecoia. If the air circulating in the blood produces only ischemia, causing transitory functional disturbances, the prognosis is good, but if primary necrosis or hemorrhages occur, they may lead to permanent irreparable damage. The author reports a case of this type. The treatment of suddenly occurring caisson disease resulting from air embolism consists in placing the patient under pressure again, administering oxygen and heat, massaging and then bringing about decompression gradually. Persons with a tendency to catarrh, disease of the eustachian tube, heart disease and various forms of hyperesthesia are not suited for caisson work, whereas persons with dry perforation of the tympanic membrane are particularly suited for it.

EDITOR'S ABSTRACT.

DEAFNESS IN OSTEOPSATHYROSIS. R. SCHNEIDER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:362 (Nov.) 1933.

Schneider discusses the syndrome of osteopsathyrosis, blue sclerotica and deafness, which has recently been recognized clinically as a triad of symptoms of hereditary nature. The osteopsathyrosis, the deafness or both may be lacking in some cases, but the symptom of blue sclerotica is practically never absent. The deafness becomes manifest at the time of puberty or earlier in the form of conduction deafness, to which a labyrinthine deafness is usually added later. The decrease in hearing is progressive; it is accompanied by severe tinnitus and sometimes ends in total deafness. The deafness is regarded as otosclerotic by most authors. The author has observed this triad of symptoms seven times in the course of one year. He calls attention to the fact that the occurrence of blue sclerotica may explain the nature of an incipient or uncharacteristic deafness or may even lead to the discovery of a disturbance in hearing before it is noticed by the patient and may thus be of significance in determining the choice of a profession.

EDITOR'S ABSTRACT.

Pharynx

PRECIPITIN REACTIONS IN THE BLOOD OF RHEUMATIC PATIENTS FOLLOWING ACUTE THROAT INFECTIONS. B. SCHLESINGER and A. G. SIGNY, Quart. J. Med. **2**:255, 1933.

Streptococcic precipitins can be demonstrated in the blood of rheumatic patients following acute streptococcic infections of the throat. Their formation is delayed until from the second to the fourth week after the onset of the nasopharyngeal infection (the end of the silent period), and their appearance in most cases foreshadows an attack of acute rheumatism. The precipitin corresponds to the type of streptococcus responsible for the infection, but a certain amount of formation of cross-precipitin may occur. The formation of precipitin is regarded as one of the manifold reactions which take place in the patient's defense mechanism during the silent period. Time is thus available for prophylactic measures if the infection of the throat has not passed unnoticed. Concentrated acetylsalicylic acid therapy during this period undoubtedly prevents serious relapses in many cases. It is not infallible, and further researches for a more reliable method are urgently required.

AUTHORS' CONCLUSIONS. [ARCH. PATH.]

TONSILLAR FUNCTION AND SURGICAL THERAPY OF THE PALATINE TONSILS. GIUSEPPE SALVADORI, Méd. inf. **2**:45 (Feb.) 1933.

One school holds that the tonsil is a lymphatic organ possessing a hematopoietic function and that its functions are closely related to, and intimately associated with, those of the spleen, thymus, bone marrow and lymphatic glands. Another school believes that it produces lymphocytes only as a defensive organ reacting to exogenous and endogenous stimuli. A third is of the belief that the tonsil represents

a residual embryonic organ, harmful to the organism in that it serves as a portal of entrance for various infections, and that it should always be removed.

Marfan concludes after an exhaustive review of the literature that the tonsils are useful organs either because of their hematopoietic function or because they serve as a front line defense against external causes of morbidity. However, when they become the seat of pathologic processes, they become harmful and should be removed, as their absence is compensated for by numerous other organs with analogous functions. COPPOLINO, Philadelphia. [AM. J. DIS. CHILD.]

PULMONARY COMPLICATIONS AFTER TONSILLECTOMY. R. WALDAPFEL, Monatschr. f. Ohrenh. **68**:143 (Feb.) 1934.

Waldapfel after a sojourn in the United States was unable to explain the relative infrequence of abscesses of the lungs after tonsillectomy reported in Germany. He thought that it might be due to the superficial narcosis with persistence of pharyngeal reflexes and to the fact that the operation is performed in the sitting position. When patients showing posttonsillectomy fever were investigated, however, he found eight in a relatively short time with various pulmonary complications which had been absent before the operation. In making the diagnosis, roentgenography was the most important factor. In all these cases the prognosis was good. The prophylaxis of pulmonary complications of tonsillectomy consists in several measures. Narcosis should be as light as possible so that the pharyngeal and cough reflexes are maintained. The operation should be as short as possible. To reduce the infection of the blood stream all possible asepsis of the oral cavity should be maintained. The author believes that the frequency of posttonsillectomy pulmonary complications in Germany is much greater than it was heretofore considered to be and that all patients showing fever after operation should be investigated with this in mind.

EDITOR'S ABSTRACT.

Larynx

SOME OBSERVATIONS ON MY THERAPY OF THE LARYNX. A. CEMACH, Monatschr. f. Ohrenh. **68**:137 (Feb.) 1934.

In two cases severe burns of the larynx were obtained by Cemach in the course of treatment for tuberculosis by direct ultraviolet irradiation. Neither of the patients had serious complications. One was practically cured, and the other was much improved. Cemach has never seen laryngeal edema following his method of irradiation. He states that it is impossible to localize the rays at one point of the larynx but that it is possible to direct them to one side or the other. In the course of the treatment uninjured areas are treated as well, but this does no harm.

EDITOR'S ABSTRACT.

Nose

ACUTE AND CHRONIC OSTEOMYELITIS FOLLOWING RESECTION OF THE SEPTUM. W. KINDLER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:345 (Nov.) 1933.

Kindler states that two personal observations and two by Glas show that submucous septum resection may be followed by osteomyelitis of the superior maxilla and the hard palate. This may assume an acute form and be attended by dangerous manifestations, such as septal abscess or necrosis of the palate, or it may take a chronic course, producing circumscribed foci of degeneration in the region of the palate process of the superior maxilla, with fistulization toward the palate, hypersensitivity in the region of the teeth and discomfort during mastication. The author thinks that the acute form of osteomyelitis and probably also the chronic form result from penetration of bacteria into the marrow spaces following a too penetrating excision of the vomer. In the chronic condition the possibility of

operative injury of the blood vessels and nerves in the nasopalatine canal and consequent necrosis of bone should also be considered. Diagnosis of the chronic condition is difficult because the symptoms appear late, which obscures their relation to the operation, and because they are localized in the region of the teeth and are mistaken for dental symptoms. Their origin may be discovered only after nasal operation and dental examination with negative results.

EDITOR'S ABSTRACT.

OSLER'S DISEASE. A. JAUERNECK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folio oto-laryng.) **25**:38, 1934.

Jauerneck describes the case of a man, aged 52, with Osler's disease in whom a severe nasal hemorrhage developed which was resistant to ordinary treatment. A large amount of a sterile coagulable animal plasma was injected into the nasal mucosa. The results were good, and the author recommends this procedure highly for all parenchymatous hemorrhages.

EDITOR'S ABSTRACT.

EXPERIMENTAL INJURY OF THE NASAL MUCOSA BY MEANS OF DUST. W. BERGER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folio oto-laryng.) **25**:60, 1934.

Berger reports the pathologic changes in the nasal mucosa, throat and larynx in twenty rabbits made to inhale various dusts and gases. Some of the animals inhaled cement dust; others, porcelain dust, pulverized white lead and oatmeal dust, and still others, carbon monoxide. The duration of inhalation was from several months to a year or more. The daily average inhalation lasted only an hour, with a dust content of from about 0.1 Gm. to 200 liters of air. In the case of carbon monoxide, a content of 0.5 per cent was used. Control examinations were made on animals not subjected to this treatment. The changes observed were nonspecific, inflammatory, irritative conditions with infiltration of the mucosa, thickening of the mucosa, erosions, fibrinous segregation, exudate formation and empyema. In addition, atrophies without stronger inflammatory processes but with empyema formation developed as the result of the severe biologic injury to the mucosa.

EDITOR'S ABSTRACT.

Miscellaneous

RESULT OF SPEECH TRAINING AFTER CLEFT PALATE OPERATION. ELSA DAVIDSON, Tr. Med.-Chir. Soc. Edinburgh, p. 23, 1933, in Edinburgh M. J., February, 1933.

Miss Davidson showed three patients with cleft palate and demonstrated the chief methods used in the training of speech. The bad habit of nasal expiration during speech must be stopped and the breath directed out through the mouth rather than through the nose. Operation for cleft palate should be performed before the child begins to speak. The movements of the soft palate should be strengthened by special exercises so as to reinforce the breathing out through the mouth. Later the children are taught the positions of the chief vowel sounds OO, OH, AW, AH, AY and EE, then the formation of the consonant sounds, their combination with vowel sounds and finally, sentences.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

PROGRESSIVE ULCERATIVE RETICULOSIS OF THE PALATE. I. S. HALL, J. Laryng. & Otol. **49**:35 (Jan.) 1934.

Hall describes the case of a woman, aged 32, with a progressive ulcer of the palate. The ulcer possessed no definite characteristics. Biopsy revealed vesiculation and edema and aggregations of cells showing mitosis and evidence of active growth. After a rapid febrile course, during which bullae developed on the bridge of the

nose, in the groin and over the thorax, the patient died. On postmortem examination, in addition to the local and cutaneous lesions septic leptomeningitis with extreme peripheral congestion and hemorrhages into the subarachnoid cavity were found. The infection had extended from the sphenoid bone through a portion of the lamina cribrosa, which was necrotic. There were pulmonary congestion and edema, and within the parenchyma there were three or four small, firm, white nodules. Similar nodules occupied the cortex of both kidneys. Histologic investigations of the nodules in the kidneys and lungs showed features similar to those in the epidermis, namely, aggregations of cells of various sizes undergoing mitosis. The author believes that death was due not directly to sloughing, hemorrhage or toxemia but to the leptomeningitis, an almost accidental complication. He reviews thirteen similar cases found in the literature. He suggests that the specific cells which were so prominent a feature of the initial ulcer and of the subsequent lesions may have originated in the reticulum cells of the lymphoid tissue of the palate. Such a conception links this ulceration with agranulocytosis, with diseases of the bone marrow and of the stroma and with lymphogranuloma. Moreover, it cannot be overlooked that the areas first affected are apt to be those which are normally the site of copious micro-organismal colonization. EDITOR'S ABSTRACT.

THE SYMPTOMATOLOGY OF THE SOFT PALATE AND ITS DIAGNOSTIC SIGNIFICANCE. O. SCHÄUFELE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:50 (Nov. 14) 1933.

After examining the soft palate in 450 nonselected patients with a wide variety of diseases, Schäufele states that yellow discoloration from hyperemia and anemia of the soft palate may appear in the most widely different diseases. This shows that these changes are not specific in the sense that one could draw from them definite conclusions as to specific diseases. The yellow discoloration of the soft palate in icterus has no practical diagnostic significance since the yellow discoloration of the sclera is a far more distinct symptom. The author was unable to confirm the opinion of some writers that yellow discoloration of the soft palate indicates a previous icterus. EDITOR'S ABSTRACT.

SPINAL FLUID IN OTOGENOUS AND RHINOGENOUS DISEASES OF THE CENTRAL NERVOUS SYSTEM. H. DEMME, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:161 (Dec. 11) 1933.

Demme found that, as a rule, the spinal fluid of a patient with otitis media or a sinus infection shows no essential changes. In some instances, however, the fluid shows an increase in the protein, especially of the globulin fraction, without clinical signs of meningitis, an increased cell count or positive cultural results. The mastic test may show all the variations from the meningitic curve to the paretic curve. Demme considers an increased globulin: albumin quotient as having a significance similar to that of the transudate in a pneumonic process. It is a danger signal, since it was possible in some patients to follow the changes in the spinal fluid from an increase in globulin to definite and fatal meningitis. The author thinks that up to the present there is no accepted explanation for the increase in globulin which occurs in some instances. EDITOR'S ABSTRACT.

GRAPHIC EXPRESSION OF TUBERCULOSIS OF THE UPPER RESPIRATORY TRACT. A. N. WOSNESSENSKY, N. A. SCHNEIDER, I. N. ALEXANDROW, J. S. SOBIN and A. P. RASPOPOW, Monatschr. f. Ohrenh. **67**:1419 (Dec.) 1933.

Wosnessensky and his collaborators describe a method for the graphic representation of tuberculosis of the upper respiratory tract. The quality of the process (productive or exudative), the form (ulcerative, infiltrative, edematous and so on), course (progressive, regressive, stationary) and the location are each indicated by an appropriate sign in the proper section of the chart.

HISTOPATHOLOGY OF SCLEROMA. B. L. FELDMANN, Monatschr. f. Ohrenh. **67**:1423 (Dec.) 1933.

Feldmann found that the classic characteristics of rhinoscleroma (Mikulicz' cells, bacteria and hyalin globules) are not always present and that their absence does not exclude a diagnosis of scleroma. Frisch's bacillus is often not found. The microscopic observation of granular, inflammatory tissue rich in blood vessels and containing numerous plasma cells and histiocytes, accompanied by obliteration of the vessels and hyalinization of the peripheral layers, is indicative of scleroma. This histologic picture, with a positive Bordet-Gengou reaction and an atrophic condition of the mucous membrane of the upper respiratory tract, may be considered characteristic of rhinoscleroma.

EDITOR'S ABSTRACT.

CORRECTION OF SADDLE NOSE WITH SPECIAL REFERENCE TO IMPLANTATION OF IVORY. G. CLAUS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:198 (Feb. 10) 1934.

Claus believes that the use of foreign materials in correcting saddle nose has advantages over that of grafts obtained from the patient. Paraffin and ivory have been used more for this purpose than most other substances. A case is described in which a correction for syphilitic saddle nose had been successfully accomplished ten years before. The patient contracted erysipelas, following which swelling of the nose persisted. Two weeks after the onset of the erysipelas a fistula developed on the bridge of the nose, through which a splinter of ivory appeared. A roentgenogram revealed a lamellated ivory graft. This was removed, and on roentgen examination the ivory appeared eroded and lamellated. Microscopic examination of the removed ivory showed giant cells along the edges, and connective tissue containing giant cells partly between the lamellae. Resorption and fissuring were also seen. With this case in mind, the author investigated the cases of fifty-one patients in whom saddle nose of various origin had been corrected by ivory implantations from 1926 to 1932. In twelve of these cases a failure resulted either because of infection or because of early expulsion of the ivory implant. In two of the remaining cases the implant had to be removed after four and six months, respectively, because of perforation of the skin by the upper end of the ivory. In eight cases the roentgenogram showed some signs of resorption of the ivory. The author concludes that the principal reason for failure in grafts which at first appear successful lies in the perforation of the skin by the upper end of the ivory. Roentgenographic examination at various intervals is a good means of determining the position of the ivory and the amount of resorption present.

EDITOR'S ABSTRACT.

OZENA OF THE TRACHEA. E. NERENZ, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:456, 1933.

Nerenz describes four cases of ozena of the trachea and gives a brief review of the previously published cases. In most of these the condition was not sharply confined to the trachea but there was concomitant or preexisting ozena of the nose. The author feels that ozena trachealis is a form of true ozena appearing in the trachea and that it has a generalized internal origin. Primary atrophy of the mucosa and the chronic infection produced thereby lead to the formation of the typical scales and the fetor. By distorting the air passage this process may endanger life. A healing tendency exists, especially after the fifth decade of life. Treatment is exceedingly difficult. Several measures are desirable. In one instance tracheotomy was necessary and was followed by recovery.

EDITOR'S ABSTRACT.

SIMULTANEOUS OCCURRENCE OF TUBERCULOSIS AND SYPHILIS. J. BECK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:472, 1933.

Beck describes three cases of apparently simultaneous development of tuberculosis and syphilis. In the first, that of a child, aged 13, biopsy on a firm infiltration of the tongue showed epithelioid cell tubercles with giant cells, so

that from a pathologic standpoint tuberculosis was diagnosed. After about nine months' treatment, in which the severe pain improved, the patient suddenly became hoarse, and the larynx showed deep infiltration. The Wassermann reaction was then three plus. Treatment with potassium iodide caused rapid improvement of the infiltration in the larynx and even some improvement of that of the tongue. In the other two cases there was evidence of the presence of both tuberculosis and syphilis. The author feels that the diagnosis of a mixed infection is often exceedingly difficult. There is some evidence that syphilis at times predisposes to tuberculosis. The histologic picture of syphilitic granulomas is often indistinguishable from that of the tuberculous lesions. A positive Wassermann reaction does not prove that a lesion is syphilitic, and a negative reaction does not rule out syphilis. In general, a tuberculous lesion is likely to be more painful than a syphilitic lesion.

EDITOR'S ABSTRACT.

EXPERIMENTAL OBSERVATIONS ON THE FUNCTION OF THE MUCOSA OF THE UPPER RESPIRATORY TRACT. A. HERRMANN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:479, 1933.

Herrmann removed the trachea from a freshly killed pig, accidentally injuring a portion by cutting out some of the cartilage. Some barium sulphate was blown down the trachea with a powder blower. Roentgenograms of this trachea taken at intervals showed clearly the action of the ciliated epithelium in pushing the particles of barium sulphate up to the larynx. This process was halted only when it reached the injured epithelium (shown also in the roentgenograms). From this point it found a new route by way of the uninjured epithelium. In dogs in a state of narcosis barium sulphate powder was introduced into the trachea in the neighborhood of the bifurcation. Here again roentgenograms showed the passage of the barium to the larynx. The author feels that defects in the ciliated epithelium of the trachea, such as tuberculous ulcers, may perhaps be shown in this way.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Feb. 5, 1934

AUSTIN A. HAYDEN, M.D., *President*

CARCINOMA OF THE LARYNX. DR. L. T. CURRY and DR. L. J. WALLNER.

We present four persons with carcinoma of the larynx from our ward in the Cook County Hospital, on three of whom laryngectomy has been performed. One patient was operated on fourteen months ago by Dr. Pearlman and Dr. Curry and one four months ago and one two and one-half months ago by Dr. Pearlman.

We present these patients for a definite reason. It is the age of "new deals" and we feel that the hoarse person needs a new deal. There is a need for publicity on hoarseness now just as there was a need for publicity to educate the public in the prophylaxis of tuberculosis a few years ago.

The salient facts as to the history and treatment of the patients presented are shown in the accompanying table. We shall designate the first patient operated on as patient 1, the next as patient 2, the next as patient 3, and the last, who was not operated on because of metastasis to the lungs, as patient 4.

In the three patients operated on the extent of the invasion by carcinoma was much greater than indirect examination of the larynx led one to suspect. All of these men had been hoarse for four months or longer before consulting a physician.

In many cases chronic hoarseness is attributed to chronic laryngitis and becomes hopelessly inoperable before cancer of the larynx is discovered. Again, one is prone to forget that a patient may have cancer, tuberculosis and syphilis in coexistence.

The problem of the otolaryngologist is to educate the public and the general practitioners and constantly to concentrate his own efforts on the seriousness of chronic hoarseness, which may be slight or variable but persistent.

In St. Clair Thomson's analysis of forty-four consecutive cases in which the original site of origin was revealed by laryngofissure and the nature of the growth verified by microscopic examination, he stated that the affected cord was mobile in twenty-seven cases; i. e., in 61 per cent of a series of proved cases there was no early symptom of immobility of the cord.

Cancer causes death in one of each nine persons who die. Of each fifty deaths from cancer, one is caused by cancer of the larynx. In about 67 per cent of cases, cancer of the larynx is intrinsic and comparatively benign for early treatment. If one can teach the patient that prolonged hoarseness is serious, there will be fewer laryngectomies and fewer deaths from cancer of the larynx. It is a sad day when a patient is compelled to choose between laryngectomy and death from asphyxiation.

DISCUSSION

DR. JOHN A. CAVANAUGH: I was impressed this evening by the number of laryngectomies, especially since they were performed by a man associated with Dr. Max Cutler. Last week I visited the tumor clinic at Hines, Ill., where Dr. Cutler showed patients with laryngeal carcinoma. He treats them with massive doses of radium and seems to believe that most growths of this nature can be cured by such treatment if seen early enough.

He stressed the point that when small doses of radium are given the tumor becomes radioresistant, and then it is difficult to obtain results.

Data on Patients Presented by Dr. Curry and Dr. Wallner

Number.....	1	2	3	4
Name.....	John M.	James S.	Harry H.	Joseph M.
Age.....	60	52	47	51
Occupation.	Laborer	Clerk, salesman	Chef	Painter
Habits.....	No smoking; chewed	Never smoked	15 cigars daily for 15 years	10-15 cigarets daily
First symptoms	Hoarseness 6 mos. before entering hospital	Hoarseness 8 mos. before entering hospital	Hoarseness 8 mos. before entering hospital	Hoarseness 6 mos. before entering hospital
Reason for hospitalization	Difficulty in breathing; choking sensation; dysphagia	Hoarseness; seen in clinic	Sore throat; 45 lb. loss in weight; hemoptysis for 5 mos.	Extreme dyspnea; dysphagia for past 3 mos.
Previous medical advice	None	None	4 mos. after onset of hoarseness; roentgen treatments	4 mos. after onset of hoarseness; after biopsy refused, laryngectomy
Indirect examination of larynx	Bilateral infiltration involving anterior two thirds of larynx; both cords fixed and much altered, with ulceration over anterior two thirds, more marked on left side; infiltration extended to ventricular bands and some swelling of arytenoids	Large ulcerating lesion involving anterior two thirds of right vocal cord; right cord fixed; left cord normal and freely movable; lesion grew rapidly after biopsy	Extensive ulceration; lesion involved both ventricles and arytenoids; cords not seen	Extensive ulcerating lesion involving right ventricular fold and arytenoid
Histologic diagnosis	Pachyderma with cells undergoing malignant change	Squamous cell carcinoma	Mixed cell carcinoma	Hornifying squamous cell carcinoma
Wassermann reaction	Negative	Negative	Negative	Negative
Roentgen examination of chest	No metastasis	No metastasis	No metastasis	Metastasis to upper lobe of right lung
Treatment:				
Entrance to hospital	Oct. 31, 1932	Sept. 7, 1933	Nov. 3, 1933	Nov. 17, 1933
Tracheotomy	Nov. 4, 1932	Sept. 15, 1933	.....	Nov. 17, 1933
Laryngectomy	Nov. 16, 1932	Sept. 27, 1933	Nov. 16, 1933	None
Local metastasis	Through thyroid cartilage, left side	Through thyroid cartilage in midline	Through thyroid cartilage in midline	Not operated on
Postoperative examination of larynx	Lesion involved anterior half of larynx but extended lower on right side subglottically	Growth extended down to level of cricoid cartilage	Large ulcerating growth extending from base of epiglottis to cricoid and involving whole anterior anterior half of larynx	Not operated on
Roentgen and radium treatment	5 roentgen treatments after laryngectomy	Radium collar 4 blocks 12.5 mg. for 48 hours, Oct. 28, 1933	15 roentgen treatments before operation	5 roentgen treatments; metastasis disappeared in chest; no hemoptysis now
Fistula	Closed spontaneously	Nassau's operation for closing old tracheal fistula tried on pharynx; small fistula persists in lower angle	Sloughed owing to previous roentgen treatments	

The slides for patients treated before and after laryngectomy were impressive. If such results can be obtained, I wonder why one should resort to mutilating surgical procedures.

Dr. A. H. Andrews: I think that the most important thing that Dr. Curry said was in regard to educating the public on the early treatment of hoarseness. This idea has not yet percolated through to the general practitioner and to the public in the way it should, and anything that can be done in this regard will be of value.

Dr. Joseph C. Beck: Dr. Curry is to be congratulated for his fine exhibition of living patients after laryngectomy. The results show that there has been progress in the development of this operation. Only a decade or so ago laryngectomy was performed by only a few surgeons, and many of their efforts were followed by death as a result of mediastinitis, pneumonia or pulmonary abscess. In contrast, today laryngectomy is a safe procedure, carrying with it a low mortality, and is being performed by many physicians throughout the country.

Irradiation therapy has been enthusiastically advocated, both high voltage roentgen irradiation after the method of Coutard and massive doses of radium. It would, of course, be ideal to produce cures by the use of these agents, but my personal experience thus far leaves a great deal to be desired.

As far as the immediate future is concerned, the hope lies in obtaining patients with an early, small intrinsic lesion that can be operated on by laryngofissure. This operation is far less mutilating and produces a fair postoperative voice, but as it is indicated in only a small percentage of cases, one must look to proper lay education in order that these patients may be seen in the early stages of the cancer, when this relatively nonmutilating operation may be employed.

Surgical Treatment of Chronic Dacryocystitis. Dr. George E. Shambaugh Jr.

Extirpation of the sac has been the generally accepted procedure for the treatment of dacryocystitis. As a substitute for this operation, intranasal dacryocystorhinostomy (the West operation) and external dacryocystorhinostomy (the Toti-Mosher operation) have been devised.

The advantages of the Toti-Mosher procedure over the others are:

1. Extirpation does not relieve tearing.

2. The Toti-Mosher operation is easier to do when excessive fibrosis makes total extirpation difficult.

3. The Toti-Mosher procedure is easier and quicker than the West operation.

4. When agger nasi ethmoid cells are present, the West operation is more difficult and less successful.

5. The scar left after the Toti-Mosher operation is negligible.

The indications for the Toti-Mosher operation are epiphora when the obstruction lies below the lacrimal sac. If doubt exists, roentgen examination after the injection of iodized poppy-seed oil 40 per cent will show the point of obstruction. In any case of chronic dacryocystitis in which extirpation is indicated the Toti-Mosher method is preferable.

Operative Procedure.—Under local anesthesia a skin incision ½ inch (1.27 cm.) long is made midway between the inner canthus and the bridge of the nose, extending down to the bone. The periosteum is elevated from the ascending process of the maxilla and lacrimal fossa, the sac thus being lifted out of its bed. The bone is removed from the lacrimal fossa, producing an opening into the ethmoid cells and the nose. The anterior ethmoid cells and the anterior third of the middle turbinate are removed, so that nothing intervenes between the septum and the open lacrimal fossa. The medial wall of the lacrimal sac is excised. The wound is closed by superficial skin sutures.

Postoperative Care.—No nasal packing or drain is necessary. The skin sutures should be removed forty-eight hours after the operation. The nose should be

kept free from adhesions and granulations until it is healed, which requires from two to three weeks.

Results.—The Toti-Mosher operation has been carried out fourteen times during the past year. There was immediate relief from symptoms in every case. In one case there was recurrence of tearing one week postoperatively owing to deficient postoperative care, which allowed granulations and adhesions to form in the nose. There were no serious postoperative complications. In one case suppuration delayed healing of the external incision for about two weeks.

PRIMARY SKIN GRAFTS IN RADICAL MASTOIDECTOMY. DR. GEORGE E. SHAMBAUGH JR.

The indication for the use of a skin graft in radical mastoidectomy is cholesteatoma uncomplicated by an extradural or perisinus abscess or by a fistula of the horizontal canal. The contraindications are acute infection of a cholesteatoma cavity by an extradural or perisinus abscess, a fistula of the horizontal canal and caries of the bone without cholesteatoma.

Operative Procedure.—The cavity produced in the radical operation is completed and the canal skin flap cut. Paraffin gauze is molded to fit the radical cavity. A half-thickness Thiersch skin graft is cut from the thigh and wrapped around the mold, especially over the facial ridge depression. The mold is inserted into the radical cavity and packed with iodoform gauze. The canal flaps are sutured back, and the postaural incision is closed.

Postoperative Care.—The skin clips or sutures should be removed on the fourth day, the packing removed from the basket on the sixth day and the basket removed on the seventh day. Sterile dressings should be kept over the canal for two weeks, and dry treatment should be employed until healing is complete, from three to four weeks.

Results.—A primary skin graft has been used four times in the past year, and the graft took each time.

Report of a Case.—On Dec. 7, 1933, radical mastoidectomy was performed on the left side. Cholesteatoma was found in the attic and aditus. A primary skin graft was employed. On December 16 the basket was removed. The graft was in situ. A little clear serum was present, but no pus. On December 28 the cavity was completely lined by skin except for a small area superiorly and posteriorly. There was no discharge. On Jan. 2, 1934, the cavity was dry. A whisper was heard in the ear operated on at 6 meters. The pars tensa and the malleus were left in place, since the ear operated on was the one with the better hearing.

DISCUSSION

DR. JOHN A. CAVANAUGH: Does Dr. Shambaugh curet out the matrix before he applies the skin graft or does he apply it only to the area devoid of matrix?

DR. GEORGE E. SHAMBAUGH JR.: The cholesteatoma matrix is not curetted out. Where there is bare bone, the graft takes; where there is matrix, the graft does not take.

TREATMENT OF IRREGULARITIES OF THE SEPTUM. DR. T. W. LEWIS.

I think that it is hardly to be questioned that submucous resection of the nasal septum has not been "sold" to the medical profession. So far as I know, the majority of the general practitioners in the country are not in favor of this procedure. Not only did I come to this conclusion from contact with general practitioners, but since the postgraduate course was established at Rush Medical College I have made it a point to ask every physician who came in concerning the general opinion in his vicinity, and that has been the almost universal report. There must be a reason. Indications for submucous resection are not, as a rule, carefully observed. And the procedure is frequently not thoroughly carried out.

There are some considerable difficulties in submucous resection which if not observed lead to failure.

The first objection, the failure to consider the indications, is rather hard to combat. Otolaryngologists often operate because of a slight irregularity of the septum that is not causing any disturbance but that offers opportunity for correction. The defect has not hindered the patient and he does not benefit by the operation. The physician who referred him to the specialist is dissatisfied with the result.

I have made it a rule that unless there is some direct indication, such as a deflection that is blocking the drainage of one or more sinuses, I leave it to the patient to decide whether the inconvenience that he suffers from the obstruction is sufficient to warrant his undergoing a rather serious operation. If the condition of his nose is not bothering him I do not insist on an operation. If there is interference with drainage as a result of the deflection, if the septum is bent over and blocks the outlet from the middle meatus or if there is infection in any sinus, whether the condition bothers him or not, he must have the operation done in order that I may adequately reach the infected sinus. Under these circumstances I insist on the operation.

Many of the members doubtless have seen many patients with insignificant defects of the septum complain bitterly of difficulty in breathing. On the other hand, I have seen patients with deflections blocking the nostril completely who were not inconvenienced. In such a case, why bother with the condition? Let it go! I present some specimens of heavy bony deflection almost at the posterior border of the vomer. Almost invariably there is a strip of cartilage extending backward from the lower border of the quadrilateral cartilage, running over the crest and passing down the posterior aspect of the deflection to be fixed in the midline. Commonly, even when the bony deflection is entirely removed, the posterior strip of cartilage is left. This, fixed in the midline, rests squarely across the nostril, holding the mucosa in exactly the position in which it was originally. Manifestly there is no relief. I have had to remove such strips of cartilage following previous operations. Another complication is injury to the perpendicular plate. Almost invariably when the septum requires operation the same injury that produces the deflection has produced thickening of the borders of the quadrilateral cartilage and the perpendicular plate, and unless that is removed there is a heavy ridge from above downward that blocks the nose. The same thing may be said of the border of the vomer, and unless that is removed no good is done.

DISCUSSION

DR. A. H. ANDREWS: My experience and observation lead me to believe that when the indications for submucous resection are clear and this is skilfully done there are few operations for chronic conditions in the nose that give greater satisfaction. It is true that great differences of opinion exist regarding this operation, but I think that the difference regarding the value of the operation is not as great as the difference in the ability of the different physicians who do the operation. When opposition to the operation is developed, it is usually due to the fact that physicians have been operating who have not mastered the technic.

DR. ROBERT SONNENSCHEIN: Dr. Lewis voiced my sentiments. I have always gone on the principle that there are only a few indications for submucous resection: (1) that the patient has subjective symptoms such as nasal obstruction; (2) that the deflection causes interference with drainage from the middle meatus, and (3) that the deflection causes headache because the middle turbinate is pressed against the lateral nasal wall.

DR. GEORGE J. MUSGRAVE: I think that there can be no valid criticism of submucous resection of the septum, a practical, safe procedure which has as its objective restoration of ventilation and drainage of the nose and accessory sinuses via the middle meatus. Submucous resection of the septum involves no destruction of the functioning tissue and in a large percentage of the cases is effective. Certainly if there is an infected accessory sinus discharging irritating secretions into the nose, with consequent intumescence of the turbinates, the procedure will

seem a failure. Such complications must be determined before operation. In this respect I have found the Proetz method of visualization and cytologic examination of the nasal discharges of great help.

DR. G. HENRY MUNDT: Submucous resection is one of the most interesting things to study in otolaryngology, for one must take into consideration the entire development of the upper jaw, in fact of the head as a whole. I think that one may study the development of the upper jaw as to depth and width and know what result will follow submucous resection.

Dr. Lewis is right in stating that there is opposition to the procedure on the part of the general practitioners. I recall an editorial in *The Journal of the American Medical Association* deprecating the opposition to nasal surgical intervention. I believe that in cases in which there is a high-arched palate with an infinitesimal decrease in the width of the nose, even though there is deflection of the septum with blocking, one can operate and produce a perfect septum and yet the patient will not have free respiration.

I was interested in another thing that Dr. Lewis said. He stated that it is necessary to do a submucous resection at times to reach an infected sinus. A thing which has been a definite indication to me for submucous resection in case of marked deflection and which I have observed frequently, is that a patient who has marked deflection usually has infection of the maxillary sinus on the side of the concavity, and there will certainly be a recurrence of the infection unless one establishes equal air passage on both sides.

DR. WILL WALTER: I am much interested in this subject and believe, as I think back over my cases, that in the ones in which there has been failure the poor result was due to: (1) operation too early in life, i. e., before ossification was complete, or (2) failure to relieve the posterior block of which Dr. Lewis spoke. As an aid in the procedure I early devised and have since used a special instrument with a flexible handle, enabling easy and free retraction of the raised mucosa to give a clear view through. This has been of great assistance to me in avoiding the second cause of failure.

DR. HOWARD C. BALLENGER: There is one objection that has not been mentioned tonight, namely, the promise of return of hearing through straightening the septum. I think that such a promise should never be made.

DR. CHARLES B. YOUNGER: Submucous resection proved a great boon to those who had experience with the Asch operation for deflection of the septum. The Asch operation was a mutilating procedure at the best. I have many times dressed wounds day after day, putting in splints and taking them out. The result was seldom satisfactory, and many times the operation resulted in permanent perforation of the septum. The submucous or window resection operation appears easy to the uninitiated but in the hands of the novice is likely to prove of doubtful benefit or an outright failure. Two of the specimens that Dr. Lewis showed illustrate well why the operation is so often unsatisfactory. In his cases there had been but a small puncture of the nasal septum which gave no relief, and the patients came to him for a second operation, with good results. If the patients are carefully selected and properly operated on the procedure is among the most satisfactory of those performed on the nose.

DR. T. W. LEWIS: As to the time of operation, I have made it a rule not to operate on young patients unless there is a very special indication for intervention, principally because of lack of cooperation on the part of the patient. A child cannot endure a long tedious operation on the nose without anesthesia, and I see no reason for subjecting a patient to the undesirable features of general anesthesia as well as to the danger of the operation. I have operated on a few children, the youngest being 7 years old. I operated on this boy twenty years or more ago because he was not developing properly. His nose was practically totally blocked as a result of injury. He was an amenable child, who allowed all sorts of manipulation of the nose. He sat for three quarters of an hour without any movement, and I succeeded in doing a very satisfactory opera-

tion. In none of the three or four patients whom I have operated on when they were young have I seen any shortening of the nose or any disturbance, so I feel that except for the factor of lack of cooperation on the part of a child there is no reason for not operating so far as age is concerned.

I handle the dislocated anterior border of the quadrilateral cartilage as follows: I reserve a strip of cartilage along the anterior border of the quadrilateral cartilage, perhaps ⅛ inch (0.3 cm.) wide. After I remove the deflected portion I strip that cartilage of the perichondrium on both sides and on its lower extremity. If the lower end of the border is thickened, as it usually is in a case of severe deflection, I cut off the lower extremity and take away the crista incisiva so that I have room to bring the strip back into the midline and into a new bed made for it. It falls back into position and is splinted on both sides. Ordinarily, I pack only one side, but in such a case I pack both sides so as to hold the strip in the new bed I have made for it. I leave the packing in position for four days, and when I take it out there is a new border in the midline.

As to headache, I have seen only one or two patients relieved by the correction of a deflection causing pressure. That sometimes happens, however.

Dr. Musgrave referred to the ineffectiveness of submucous resection to relieve disturbances of the sinuses. Sinusitis is not relieved by submucous resection. One must treat the sinus after the resection is properly done. The operation is performed in order to reach the sinus adequately and at the same time insure free drainage.

PATHOLOGY OF OTOSCLEROSIS. DR. E. W. HAGENS.

A discussion of the pathologic process of otosclerosis usually brings to mind the changes found in the bony capsule of the labyrinth. These are the changes most often seen, although alterations in the cochlea and the eighth nerve have been found.

The characteristic feature of the pathologic process in the bone is that it occurs in localized patches or foci. The focus usually begins in the enchondral bone, and as it enlarges it involves either the endosteal or the periosteal layer or both. The focus is more vascular than normal bone except in the oldest foci, which are sclerotic. In the early and middle stages marrow-like cavities are often prominent. Wide, thin-walled vessels are found in the cavities. Occasionally a large vessel may be seen connecting the otosclerotic bone with the adjacent uninvaded bone. In the marrow-like cavities are also found connective tissue cells, some resembling fibroblasts and others, more spherical, which are difficult to identify. The bone cells of the focus rarely have the haversian configuration, being scattered or clumped together haphazardly. Some of the cells are recognized easily and others with difficulty. In the vascular foci a fair number of osteoclasts and osteoblasts are sometimes seen. The latter are situated at the bony edges lining the spaces, and frequently a pink-staining layer of new bone is noted just beneath. It seems possible that if this bone-building process were to keep up for several years the tissue would gradually become more dense and the spaces smaller, until finally a dense tissue would result.

Some bones show only one focus and others more, as many as four or even six. The condition is frequently bilateral. The most common site for a focus is in the region between the cochlea, vestibule and foot plate of the stapes and the processus cochleariformis. The bone of this region is often peculiar. In some cases remnants of cartilage are prominent. The term Cozzolino's zone is often applied to the fissula ante fenestram, which is found in this area. Recently Bast found otosclerosis surrounding the connective tissue of the fissula, and he emphasized the fact that the fissula instead of being the starting point for the focus, must be invaded secondarily. Certainly the stapes and its foot plate are involved only secondarily. The sites of other foci are: about the chochlea, around and filling in the round window and about the canals and vestibule. When otosclerosis has been found associated with suppurative otitis media it has seemed to me that there was no demonstrable relation between the two condi-

tions. In cases in which otitis was not present and there was no history of otitis and in which unilateral otosclerosis was revealed on postmortem examination, it was evident that in the otosclerotic ear there was a definite increase in the vascularity of the subepithelial tissue overlying the stapedial and the adjacent promontory region. However, no evidence of infection, as evidenced by the presence of round or plasma cells, was found. The vascularity seen could easily explain the finding of Schwartze's sign, which is often noted clinically in these cases. It may be possible, however, that a previous infection had taken place without leaving permanent changes, the infection having resolved itself so that the microscope was unable to reveal what had gone before.

Numerous ideas have been put forth as to (1) the causal factors and (2) the development of the bony foci. With regard to the causal factors, it is known that the condition frequently occurs in families and also that females are more frequently attacked than males. So far as I know, the youngest child in whom foci have been found was about 1 year of age. Recently Guggenheim reported the presence of otosclerosis in a 7 months fetus. Full details of the case are still to be reported. This speaks in favor of the theory of a hereditary condition as against that of a postnatal, or acquired, condition. In the embryonic ears that I have sectioned, no evidence of foci has been seen. Some observers have expressed the belief that in these cases there is an inherent defect of the ear mechanism or a congenital inferiority of the ear. Such a point has been difficult to demonstrate because it has been impossible to show changes microscopically to support it. Other observers hold that the foci develop from remnants of embryonic tissue. Still others point to a strain or fracture in Cozzolino's zone.

A point of interest is the possibility that the foci develop from cartilaginous remnants. Several of the foci in my sections appeared to be closely related to or actually touching the remnants. However, cartilage remnants are found in regions in which foci are found rarely, if at all, and remnants have been seen in many adult temporal bones showing no foci. Thus the importance of the relationship between such remnants and otosclerotic foci is not easily settled. It has been said that perhaps the foci develop where ossification occurred last. The sections of the ears of embryos and of new-born and older infants that I have examined have shown that regions which are frequently cartilaginous when all the surrounding tissue is ossified are, in addition to Cozzolino's zone, the regions behind the vestibule in the posterolateral and posteromedial portions of the petrous bone.

Various ideas have been expressed as to what may incite the development of the foci, such as constriction of the vessels in the petrous bone, stasis of these vessels, chronic local inflammation and chronic local infection. Among several factors influencing the condition may be mentioned: gastro-intestinal toxemia, any focus of infection, syphilis, pregnancy, a disturbance of the glands of internal secretion, a vascular disturbance, tuberculosis, a faulty diet (lacking in vitamins) and blue sclera with fragile bones.

As to the development of the foci once they are started, there are two main ideas. The first, and perhaps the one most frequently mentioned, is that there is absorption of normal bone followed by replacement by new, vascular bone, and that later this bone becomes denser. The second view is that the focus is a tumor-like formation which destroys the normal bone as it develops.

Several points may be discussed concerning the first view. One would expect to find evidence of absorption of the normal bone in the region of the focus. Yet such evidence is definitely lacking in some foci and indistinct in others. In the sections that I have seen showing possible changes, the bone adjacent to the focus appeared rarefied. In the absence of osteoclasts, etc., the most likely view is that the alteration was the result of some chemical action.

The view that the focus is tumor-like has seemed plausible because of the frequent lack of evidence of alteration of bone near the border of the focus. However, the foci do not simulate ordinary tumors in that they do not have a

tumor cell common to them. The most characteristic evidence of advance in my sections seemed to be the finger-like vascular projections extending from the focus into the adjacent bone. In some instances these were covered by several rows of blue-staining cells with canaliculi.

Thus the focus could enlarge by gradually throwing out tentacles into the adjacent bone and slowly replacing it. The adjacent bone would be prepared, probably by a chemical action. The enlargement of the foot plate of the stapes indicates that the condition not only replaces previously existing bone but develops beyond the previous bounds, thus simulating hyperostosis.

DISCUSSION

DR. J. GORDON WILSON: Dr. Hagens has given an excellent demonstration. The origin of otosclerosis is still unsettled, and its pathology and pathogenesis offer an attractive field for investigation. The disease presents, in a unique bone, a localized lesion which may have a profound effect on hearing. The facts that an otosclerotic nodule can stop growing and that the tempo of its growth may vary in the same person justify the hope that what nature can do man may also accomplish.

Cozzolino's zone, to which reference has been made, lies around a small anterior fissure which lies in the labyrinth capsule between the stapes and the cochlea. It is a medley of bone, cartilage and fibrous tissue persisting in varying degree into adult life. It is almost always included more or less when otosclerosis develops at the seat of election near the stapes.

To help dispel the fog which surrounds the pathogenesis of otosclerosis, painstaking work such as Dr. Hagens is doing must go forward. It is encouraging to see the younger men at Rush Medical College keeping up the traditions of the school.

DR. FRANCIS L. LEDERER: One cannot but be impressed, from an observation of these lantern slides, with the similarity of the process to that of osteosclerosis. In the first and second cases there was a history of aural suppuration over a period of many years, and one is, therefore, struck by the possibility that the process was osteosclerosis following suppuration. The changes shown in Dr. Hagens' preparations of otosclerotic bone differ materially from the classic histologic picture of this entity, wherein no demonstrable inflammatory process, much less one of a destructive nature, is seen. It seems to me that while histologically he has shown the process to be otosclerosis I should have experienced great difficulty in making the diagnosis clinically as was done in these cases. If it is true that Guggenheim can demonstrate otosclerotic plaques in a 7 months fetus, one will have to discard more than half of the existing theories of etiologic factors which have been suggested and think more strongly of the possibility that the foci develop from remnants of embryonic tissue.

DR. ALFRED LEWY: Has a report of Dr. Guggenheim's case in a 7 months fetus been published?

DR. E. W. HAGENS: I thank Dr. Wilson for his discussion. In regard to osteoclasts, I found them in the second and third cases; they were not abundant but were present in sufficient numbers to be detected. The point that struck me was that I found the osteoclasts and osteoblasts in the focus and not at the border, as is reported in the literature. My impression of Bast's article and work was that he found the focus in that territory and it seemed to have surrounded the fissula ante fenestram, and he thought the fissula had not been the starting point; but I agree with Dr. Wilson that the fissula becomes included.

DR. J. GORDON WILSON: I think that Bast was working toward just what Dr. Hagens showed. The latter demonstrated that the fissula was very evident, and it never was obliterated in the cases described by the former.

DR. E. W. HAGENS: Dr. Lederer wondered whether the process might not be osteosclerosis with infection. I have seen in the petrosa bone building occurring simultaneously with destruction even in acute cases, and certainly it is

possible for osteosclerosis to proceed in this way. However, in the specimens that I presented the staining reaction is different from that of osteosclerosis and the location is also different, and I cannot see how any one can become confused between osteosclerosis and otosclerosis. I think that if one studies the sections one will see that the picture corresponds in every way with the classic description that has been given. The foci are in the typical sites and have the same staining reaction, and the structure is almost similar to that which has been described.

Dr. Guggenheim's article was published briefly in the *Annals of Otology, Rhinology and Laryngology* (**42**:690 [Sept.] 1933). The detailed report has not yet been published.

OBSTACLES ENCOUNTERED IN ELECTROCOAGULATION OF THE TONSILS. DR. WILLIAM J. YONKER.

My associates and I were in an attitude of mind to expect favorable results from electrocoagulation of the tonsils. The reason for this was the spreading of propaganda by radio, by clinics provided by manufacturers and by the reports in certain medical publications and in the general press. So we undertook to give the method an unbiased investigation.

Several members of our staff investigated the procedure in the clinic and in their private practice. We have used various types of machines. We have watched operators demonstrate the method in clinics given for that purpose and observed some utilizing the method in their private practice. We have had physicians limiting themselves to physical therapy assist us in certain cases and have had representatives of manufacturers of apparatus for physical therapy advise us as to the proper use of the machines.

Our results have been unsatisfactory, and we are still in doubt as to whether there are any indications for this method of removal of tonsils. In the *Surgical Clinics of North America* (**12**:1157 [Oct.] 1932) Drs. G. E. Shambaugh and C. L. Dougherty and I reported our experiences with electrocoagulation in the practice of otolaryngology as it was investigated in the department of otolaryngology of Rush Medical College. Since that report we have continued to follow up the patients and find no reason for altering the conclusions there expressed.

Among the difficulties connected with this method may be mentioned the pain occurring during and after the treatments, the length of time that a patient must be under treatment, causing an unnecessary delay in the removal of pathologic tonsils, the complication of secondary bleeding, and especially the uncertainty as to whether all pathologic tissue has been removed. In the series of cases that we are reporting now we are not satisfied that all tonsillar tissue has been removed in a single case. Moreover, we have had opportunity in the clinic of examining patients who have had electrocoagulation done elsewhere, and in not a single instance have we found that the tonsils have been eradicated.

Contrary to what is often affirmed, pain is rather prominent both before and after treatment by electrocoagulation. By topical applications of anesthetic solutions, such as cocaine, nupercaine and butyn, one cannot render the throat insensitive to this treatment. We have attended a clinic by an electrocoagulation enthusiast who first injected a solution of nupercaine about the tonsils, dissected them free anteriorly and finally plunged a needle through various parts of the tonsil in an effort to eradicate it in one treatment; but one hesitates to approve such radical procedures. Indeed, since water-logged tissues tend to diffuse electrical currents, we should hesitate to inject anesthetic fluids about tonsils previous to electrocoagulation because of the fear of damaging the tissues about the tonsils, particularly the larger vessels. It is true that the patient does not note much pain during the early treatments when one confines the process to the tonsillar tissue and remains at a considerable distance from the pillars. The nearer that one approaches the capsule, however, the more pronounced is the pain. Besides the pain experienced, the patient is disturbed by the noise of the machine, the increased salivation and especially the gagging.

The pain following the treatment varies with the sensitiveness of the patient, the thoroughness of the treatment and the nearness of the approach to the so-called tonsillar capsule. In case 15, the patient noted pain for four days following each treatment; in case 6, pain was sometimes felt as long as seven or eight days following an application; and in case 7 the discomfort complained of by the patient and the inflammatory reactions noted by the physician following only one application were fully equivalent to the discomfort experienced following surgical enucleation. If one bears in mind that patients must be treated from twelve to twenty-four times or more before one can hope to obliterate the tonsils, it will readily be realized that the total amount of suffering experienced by the patient is significant.

Another obstacle encountered is the length of time required to complete the series of treatments. If a patient has both tonsils treated at one time, comes in regularly once a week and requires only twelve treatments for complete obliteration, three months will be needed to complete the series of treatments. If one side is treated at one sitting, the patient will have to come regularly for from two to six months or more. The method requires a great amount of time of both the patient and the physician.

This method unduly delays the removal of diseased tonsils. When one can trace an ailment, such as endocarditis, nephritis, acute inflammatory rheumatism or iritis, to diseased tonsils, it is far better to remove this menace to health in one sitting than by a long series of treatments. In case 13, the patient had an attack of acute follicular tonsillitis following the first treatment, which could have been prevented if the tonsils had been enucleated by the usual surgical procedure.

It has been argued that the initial treatments by electrocoagulation tend to sterilize the tonsils, a proposition that we are not willing to grant. Infection in tonsillar tissue may be deep-seated. Crypts in the tonsils may be scarred over as a result of these treatments, or they may be so deep that they are not reached by the first treatments. In case 9 attacks of tonsillitis occurred even after twenty-four treatments had been given.

The fear of bleeding induces many patients and some physicians to look favorably on electrocoagulation, but it must not be forgotten that this may be a complication of electrocoagulation. To the argument that this complication is a result of overzealous treatment, one may reply that bleeding following surgical removal of the tonsils is also usually a result of some indiscretion in technic. That bleeding may occur as a result of electrocoagulation is seen from case 15 and case 17. We submit that it is easier to control secondary bleeding in tissue that is healthy than in tissue that is partly coagulated and possibly friable.

The most serious obstacle which we have encountered in electrocoagulation of tonsils is the extreme difficulty of eradicating all diseased tissue by this method. In case 16 ten treatments were given to each tonsil, in case 6 nine treatments and in case 9 twenty-four treatments, and still all the tonsillar tissue was not removed. The reason for this difficulty is that as one approaches the so-called capsule it is hard to distinguish between scar tissue, fibrous lymphoid tissue and lymphoid tissue embedded or covered by scar tissue. Sometimes the base of the fossa appears mouse-eaten, and one wonders whether the elevations are lymphoid tissue embedded in scar tissue or whether the depressions are cavities in the underlying muscle tissue.

We may continue to experiment with electrocoagulation, but because of the various obstacles mentioned our experience with this method of removing tonsils has thus far been disappointing.

Seventeen cases are reported.

DISCUSSION

DR. A. R. HOLLENDER: No one will dispute the fact that electrocoagulation is unsuitable as a routine method for removal of the faucial tonsils. I have emphasized this in my writings and on many other occasions. I believe, how-

ever, that electrosurgery has a definite place in tonsillar therapy, particularly in selected cases which do not meet the usual indications. I agree with the essayist that the procedure is somewhat painful at the time it is carried out and also that there is some local reaction subsequently, but these facts do not necessarily detract from the value of the method. I do not agree with the essayist that it is not possible to extirpate all the tonsillar tissue so far as can be determined with the naked eye. One has observed the extirpation of large cancerous growths by electrocoagulation. Is there any reason why it cannot destroy benign tissue? The whole question becomes, then, not whether it is possible to eradicate the tonsillar tissue but whether one can determine when all the tonsillar tissue is removed. Does not this question arise with the present surgical methods of removal?

It is unfortunate that laryngologists who have never used electrocoagulation and are totally unfamiliar with it are ready promptly to condemn it under any and all circumstances. It appears to me that a more considerate point of view is in order, so that one may not be too hasty in becoming prejudiced against a method which in properly trained hands possesses merit and which adds to the available methods of therapy of the tonsils. The laryngologist should train himself in electrosurgery and should withhold judgment until by his own experience he has qualified himself to pass an unbiased opinion.

Dr. Harry L. Pollock: I fully agree with the essayist that there is absolutely no indication, with one exception, for removal of tonsils by electrocoagulation. So far as physical objection to surgical removal is concerned, I think there is none. There is no patient who presents himself—I do not care what the blood pressure is or how severe diabetes he has—who cannot be treated by surgical intervention after the conditions have been met. The only justification for removal of the tonsils by electrocoagulation, in my opinion, is to satisfy the patient who demands electrocoagulation. In my experience with electrocoagulation of the tonsils, in only one patient did I feel that the tonsils were entirely removed, and that patient required about sixty treatments. It took between a year and eighteen months to complete the treatment, but the patient was willing to give the time and I was willing to do the work. Most of the cases resemble those shown tonight. When one gets close to the capsule one does not wish to go too deep and produce a perforation or a slough and so is reluctant to go far enough to remove the tonsillar tissue entirely. In one case I thought that removal was complete with satisfactory granulation tissue, but the patient returned three months later with acute tonsillitis. Behind the pillar it is difficult to remove all the tissue. I agree with the essayist in all other respects. It seems to me that in the clinics where these cases are demonstrated only the first or second application is shown. About two years ago I went to one of the clinics which advertised demonstrations of complete removal of the tonsils, and when I arrived I was told that I was just too late to see the patients who had complete removal.

In my opinion there is no physical condition that justifies the use of electrocoagulation in the removal of the tonsils, its use being justified only for patients who refuse surgical intervention and are willing to put up with the long time and excessive expense of the method. In such cases, for psychologic reasons, one must resort to this method.

Dr. John F. Delph: Has Dr. Yonker's experience been the same with tonsillar tags?

Dr. Robert Sonnenschein: Three or four years ago at one of the meetings of the Middle Section of the Triological Society this subject was discussed, and at that time I stated practically what Dr. Yonker said with reference to the inefficacy and undesirability of attempting to remove tonsils by electrocoagulation. I think that the point which Dr. Pollock made about the psychology of the patient is true. There is a certain mystery in the phrase "removing the tonsil by electricity." When this method was heralded naturally many patients insisted on having it used. My associates and I had to get a machine. We tried it but did not advise its use for the reasons mentioned by Dr. Yonker.

So far as tags are concerned, I think that there is some indication for the use of electrocoagulation for patients presenting them. Before this method was devised, we used the electrocautery for this purpose, and we still do, placing a little powdered cocaine on the tag, plunging the cautery point into the tissue and destroying it.

So far as secondary hemorrhage is concerned, we were told by the manufacturers of the apparatus that coagulation prevents hemorrhage, but one of the worst cases of hemorrhage that I have seen followed the use of this method. Two applications were made, and each time on the ninth day thereafter a terrific hemorrhage occurred which required through-and-through suture for control. I believe that surgical intervention is the best method for removing tonsils.

DR. ELLISON L. ROSS, Waukegan, Ill.: I cannot add anything to Dr. Yonker's statement. He stated his findings clearly and completely. All I could say would be a repetition of his observations and conclusions.

DR. HOWARD C. BALLENGER: About six months ago I saw one patient on whom complete tonsillectomy had apparently been done by diathermy, and I congratulated the patient on having smooth, clean fossae. Later she came into the office with a sore throat, and I detected a small bulging in the bed of the tonsillar fossa on the right side. I incised this and obtained pus and underneath found a piece of tonsil completely bound under the smooth scar.

DR. FRANK J. NOVAK JR.: About ten years ago I called attention to the possibility of sealing in a diseased crypt by diathermy so effectively that even careful examination would not reveal the possibility that the scar concealed diseased tonsil beneath it. Succeeding years have brought to light cases in which this had occurred, to the great detriment of the patients. I agree entirely with Dr. Yonker in his conclusions.

DR. A. H. ANDREWS: A study of the literature on this subject reveals that one of the early writers was a former member of this Society—the late Dr. Edwin Pynchon. He undertook to treat diseased tonsils by cauterization of the crypts, with the result that many of them were sealed and later had to be opened surgically. He also removed tonsils with the cautery, and while the operation seemed to be satisfactory to him the patients complained bitterly of pain both during and for several days after the operation.

I am willing to go on record as believing that there is something in the method of coagulation. The physician who uses it without having seen some of the work of operators who have had experience with it is hardly justified in condemning the method because he has not found it satisfactory in his own hands.

DR. W. J. YONKER: Replying to Dr. Hollender, we do not say that all tonsillar tissue cannot be removed by this method, but we wish to stress the extreme difficulty of doing so.

Dr. Pollock stated that electrocoagulation may possibly be indicated when a patient insists on having his tonsils removed by this method, refusing to submit to the usual operation. We have found that patients usually change their minds after a few treatments. It is better not to treat patients at all by this method unless they submit to the entire series of treatments, for a few treatments may seal in infection.

A question was asked regarding the use of electrocoagulation for tonsillar tags. Our position is that a superficial tag is rather easily removed by this method but that such tags are usually no menace to health. The larger, deep-seated tags are removed with as great a difficulty as are tonsils.

DIATHERMY IN THE TREATMENT OF SOME CONDITIONS OF THE NOSE AND THROAT OTHER THAN DISEASE OF THE TONSILS. DR. C. L. DOUGHERTY.

Diathermy seems, *a priori*, to be well suited for the treatment of a variety of nasal conditions. For example, nasal alterations which are usually associated with an allergic state suggest at once the possibility of influencing the allergy clinically

by its use. There are several alterations in the nasal mucosa that are recognized as more or less typical of an allergic condition. These conditions are grouped as, or associated with, what has been ordinarily described as hyperesthetic rhinitis and hyperplastic conditions of the ethmoid labyrinth. Hyperesthetic rhinitis seems to be suitable for treatment by diathermy, the electric current used being considerably below the actual cauterization or even coagulation point and just sufficient to raise the temperature of the tissues a few degrees above the normal. The use of diathermy in such cases requires considerable careful study and observation before it is possible to determine just what strength of current and what length of application are required to bring about the desired results.

I have applied diathermy in a sufficient number of cases of hyperesthetic rhinitis and have observed the clinical phenomena carefully over a sufficiently long period to enable me to form a favorable opinion as to its usefulness. While the results seem favorable, I do not feel that I should say more than that I am encouraged to pursue the method further and study its effects as to permanency and perhaps as to the untoward results which may develop later. The results, although most encouraging, do not warrant a more definite statement at this time.

Regarding the condition in the region of the ethmoid labyrinth so frequently associated with allergy, namely, hyperplastic ethmoiditis, I have endeavored to remain open-minded about the usefulness of diathermy. Certain facts are at once appreciated. It does not seem proper to use diathermy in the cautery strength or actually to coagulate tissue in the ethmoid region for reasons that are obvious. These structures lie altogether too close to the intracranial cavity to permit too vigorous application of the electric current in this region. In this connection, I should report that I have applied the diathermy current in cases of hyperplasia of the ethmoid region too cautiously to make certain of any favorable and lasting results. So far no appreciable alterations resulting from this form of treatment have been observed in the ethmoid region. These are the two outstanding intranasal conditions which seem suitable for favorable influence by diathermy.

As for diathermy in nasal conditions other than those just cited, its use can be recommended to destroy bleeding vessels in the nasal septum and varices in the nose, pharynx, larynx and base of the tongue. The apparent absence of gross scarring seems to be a desirable feature.

Hypertrophy of the turbinates is favorably influenced and reduced by the use of diathermy. This procedure has been used in treating the commonly found mulberry hypertrophy of the posterior end of the inferior turbinate, and the results warrant the conclusion that such mulberry hypertrophy may be more simply, easily and conveniently handled by diathermy than with the snare.

Discrete areas of hyperplastic lymphoid tissue occurring on the walls of the pharynx and nasopharynx, as well as those sometimes found in the tonsillar fossae following tonsillectomy, are simply and easily removed by means of diathermy. In a limited number of cases in which a portion of redundant tissue remains after a submucous resection of the nasal septum diathermy has been used satisfactorily.

Experiments are now under way to measure the actual amount of heat used in the tissues, which will definitely establish the optimum conditions for the application of diathermy. These will be reported on later.

DISCUSSION

DR. T. C. GALLOWAY: I have seen some striking results in cases of allergic conditions of the nose. In one case the allergist and I had been unable to do anything, and as a last measure I removed the polyps and coagulated the base of the polyps and the polypoid middle turbinate. The woman had had complete nasal block and had been asthmatic, and the polyps had been recurring very quickly. She came back in two months with a clean nose and no allergic symptoms. I think that this treatment, possibly by interruption of a reflex arc, acted somewhat like Dr. Otto Stein's injection of alcohol and Dr. Vail's use of trichloracetic acid, the results of which are occasionally striking.

Regular Meeting, March 5, 1934

AUSTIN A. HAYDEN, M.D., *President, in the Chair*

DIATHERMY AND RADIUM IN TREATMENT OF MALIGNANT GROWTHS; COMBINED LARYNGOFISSURE FOR CANCER OF THE LARYNX. DR. T. C. GALLOWAY and DR. M. T. LAMPERT.

A brief résumé of the methods of treating cancer of the larynx with a statistical analysis of their results and advantages is made. A combined method of treatment by laryngofissure, electrocoagulation and radiation is proposed. Two cases are cited in which the lesion was exposed by laryingofissure and partly excised, the base as well as any suspicious tissue was treated by diathermy, wide excision of the neighboring thyroid cartilage was made and ten needles of radium (2 mg. each) were laid as a plaque to be removed after seventy-two hours. The method is recommended as a substitute for the more mutilating laryngectomy.

Illustrations are presented to show the results of diathermy alone or combined with radium and roentgen therapy including: a trap-door approach through the nasal bridge for adenocarcinoma filling the nose and nasal sinuses; carcinoma of the alveolar ridge and antrum; extensive squamous cell carcinoma of the preauricular region; removal of the epiglottis by diathermy snare after the application of radium, and a case of twenty carcinomas of the face, lids, neck and arms, including basal cell, cylindroma and squamous cell types.

DISCUSSION

DR. A. JAMES LARKIN: There is a possible misapprehension regarding the term radiosensitivity. Radiosensitivity is apparently quantitative; that is, tumors called radiosensitive are destroyed with relatively small amounts of radium, e. g., 3 erythema doses, and those that are radioresistant may be destroyed with larger amounts of radium, e. g., from 8 to 12 erythema doses. The destroying of malignant growths by irradiation is a question of technic. The dosage required to destroy certain types of lesions according to their microscopic appearance is pretty well known. Irradiation through normal structures to reach the pathologic tissue is inadequate, except in the case of extremely sensitive lympho-epithelial lesions or of the transitional cell epithelioma, usually found in the pharynx. Coutard's results are excellent in the pharynx, where the lesions are usually radiosensitive. More resistant lesions require higher amounts of radiation than can be given by external treatment.

I think that progress in radiation treatment of these conditions is being made, but it requires the cooperation of men of the authors' type who are working in the field and of those who work with radiation. The radiologists are not dexterous in the application, and they are not at home in the field, but they do know something about general principles. Therefore, I urge that those practicing the two specialties try to work together. The burden of the otolaryngologists is the greater, but we radiologists will try to do all that we can to help.

DR. FRANCIS L. LEDERER: It is perhaps unnecessary to remark that any work along the lines Dr. Galloway and Dr. Lampert presented represents a great deal of labor. To try to reduce the matter of malignant growth to a mathematical formula, or in certain cases to try to stress definite indications for a certain operation, may be extremely difficult. Furthermore, to rely on microscopic evidence as a criterion is often inadequate. The microscope may show invasion of structures involvement of which was not anticipated on visual examination. The degree of malignancy, type of neoplasm, presence or absence of metastases, fixation of the growth, anatomic relations and location, direction of growth, extent and nature of previous therapy and condition of the patient are all to be evaluated. As to the remark that laryngectomy is a terrifying experience, I can think of nothing more terrifying than the disease itself. Window resection, or laryngofissure, Dr. Lampert said, can be carried out, and if it does not suffice and there is a recurrence,

laryngectomy may still be performed. I think it has gradually come to be felt by otolaryngologists that the patient does not present a favorable condition for this operation when in the window resection a natural barrier is removed. If this is the essayists' feeling, that the operative intervention is not complete enough to remove all of the cellular elements, surely there is a definite disadvantage to it. Why not do the most complete procedure, laryngectomy?

Perhaps when one summarizes the experience in the field, it may be thought that the entire attack is not one with roentgen rays, radium, surgery or diathermy alone but, as Dr. Galloway expressed it, perhaps a combined attack with all these measures. Certainly, while the microscope has aided considerably, there is, as Dr. Larkin suggested, a tremendous field open for investigation. The cellular features of malignant growth are stressed today more and more as therapeutic indications, but they must be considered with reservations.

Dr. Joseph C. Beck: There is not much to discuss, except that one could report one's own sad experiences. Dr. Galloway showed a slide illustrating his early results with irradiation, and one can show early results with surgical intervention; but such results are not obtained now with surgical treatment of the larynx or with any other kind of treatment.

I did not find that the authors laid enough stress on glandular metastases. The presence of involved glands in the neck makes a tumor absolutely inoperable. Even though one may resort to block resection, one sees recurrences in the neck even with the best interstitial and distant irradiation, either with roentgen rays or with radium, and it has been my experience to see those patients die ultimately from general carcinomatosis.

Dr. Galloway may properly be called a pioneer in the use of endothermic treatment. He has been working for a long time on this subject. Recurrences from necrosis and other things may discourage one, but I think that as one goes along one finds that this type of treatment, particularly for the epidermal type of tumors in the tongue, palate and upper jaw, gives splendid results and a cure that lasts a number of years. Therefore, the method of electrotherapy is highly commendable. I saw the patients this evening and studied the microscopic sections and was particularly interested in the case in which a carcinoma of the epiglottis was treated by irradiation and a section was subsequently removed and no recurrence found. The man has epitheliitis, but there is no recurrence of the growth. It is unusual to find carcinoma confined to the epiglottis, and I am pleased to see this case.

Dr. T. C. Galloway: We are fortunate that Dr. Larkin is now working with us as radiologists at the Cook County Hospital and giving us the advantage of his experience. I think, as has been said, that such growths need the combination of all effective measures, and with both diathermy and surgical measures we are now getting some place, where as previously the situation was rather hopeless.

Dr. Beck is right about the glands. Block resection has not been successful in my experience; however, irradiation is not extremely successful either.

Endoscopic Experience. Dr. John F. Delph.

I am sure that every one who does endoscopic work at some time or other encounters cases which are interesting or unusual. Tonight I wish to present four cases encountered during the past few years which I consider worth reporting.

The first case is one of a foreign body in the esophagus. On looking at the film, one can see a large spoon held high in the esophagus. It is an ordinary army issue teaspoon, bent double and forced into the esophagus by an insane person. Removal by the oral route was impossible, so it was forced into the stomach and subsequently removed by gastroscopy. During the procedure the esophagus was torn only slightly, and recovery was uneventful. This case is of interest chiefly because of the large size of the object and because of the proof it affords of the elasticity of the esophagus.

The second case is that of a man, aged 56, who had a history of loss of weight of 25 pounds (11.3 Kg.) and recurring pleurisy over a period of five years. There was definite blockage of the right bronchus, iodized poppy-seed oil 40 per

cent stopping short after leaving the trachea. The physical findings were in keeping with a blocked bronchus. At bronchoscopic examination a mass was found occluding the bronchus of the lower lobe of the right lung. Pieces removed showed a mass composed of closely packed polygonal and spindle-shaped cells with occasional mitoses. Between these cells was a loose-meshed stroma composed of connective tissue cells and infiltrated with leukocytes. The cells showed a definite radial arrangement about thin-walled blood vessels. A diagnosis of perivascular hemangio-endothelioma was made, and subsequently five radon seeds were implanted in the mass. In three months the patient had gained over 50 pounds (23 Kg.), the lower part of the pulmonary field had opened up, and lipiodol was seen in the bronchiectatic spaces. Exactly one year later the patient reentered the hospital with a return of the old symptoms. Death occurred in a few days. Autopsy showed a large squamous cell carcinoma originating in the right bronchus with multiple metastases into the lung, pericardium, superior vena cava and esophagus.

The third case is that of a man, aged 53, who complained of pain in the right side of the chest. Roentgen examination of the chest after injection of iodized poppy-seed oil 40 per cent showed definite blockage of the right main bronchus. At bronchoscopic examination a yellowish mass was found occluding the main bronchus. Portions of the mass came away with the bronchoscope, and several pieces were removed with a forceps. They were soft and gelatinous, and histologic section showed them to be composed of a myxomatous vascular tissue similar in structure to a nasal polyp. There were a few isolated masses of stratified squamous epithelium, and a few mitoses were found. The general appearance was that of an inflammatory process. A few months later the patient died from extensive carcinomatosis.

The second and third cases are presented to show the difficulty of obtaining good biopsy specimens through the bronchoscope. No doubt carcinoma was present at the time, but it is difficult and dangerous to bite out enough material to show all cell structures. Fatal hemorrhage has sometimes occurred. In the second case, the growth histologically looked like a radiosensitive tumor, and for a while one was misled. In the second case the tissue no doubt was that produced by pressure and edema.

The fourth case is that of a 4 year old child who had a history of aspirating a pistachio nut two months before I saw him. Since then he had had a cough and had been more or less croupous. On looking at the films, one sees a left emphysematous lung and a compressed right lung with the heart and mediastinum pushed to the right. A diagnosis was made of a foreign body in the left lung producing obstructive emphysema due to a ball-valve action. At bronchoscopy the pulmonary field was only moderately filled with secretion and did not show the picture produced by a vegetable foreign body. No foreign body was found, but following aspiration the heart and mediastinum moved back to the midline, and the child has been free from symptoms since.

I present this case because it is the third time that I have had a similar experience, in each case the symptoms being caused by a peanut and the patient recovering after one aspiration. To offer explanations would be pure conjecture. It is hardly probable that a mucous plug could cause such obstruction for so long a time (two months).

DISCUSSION

DR. T. C. GALLOWAY: I have two slides that I should like to show because they represent the unexpected finding of a foreign body.

The first slide is for a patient who came into the Children's Hospital with a boil on the buttock. The boil was opened, but fever persisted. A roentgenogram of the chest showed plugging in the entire pulmonary field. Bronchoscopy was done, and a peanut was removed; the child recovered.

The second slide bears on some interesting work that is being done by Dr. Anspach and Dr. Brennemann in relation to bronchiectasis. In going back over old cases, they found this film showing a shadow. There was no history of a foreign body, but a bronchoscopy was done, and a peanut was found in the terminal

bronchus on the right side. This was responsible for the atelectasis which the patient presented, and Drs. Anspach and Brennemann believe that it would inevitably have led to bronchiectasis.

DR. ROBERT GOOD: On the subject of surprising experience in bronchoscopy, I have three cases to add. One was that of a woman who while eating soup felt "something go into the lung." A roentgenogram showed nothing. Her temperature was about 102 F. for five or six days, so I did a bronchoscopy. Near the pleura I found a bronchiole which contained pus. I aspirated it but did not obtain a foreign body. The patient was much chagrined, and I suggested taking her to Dr. Jackson in Philadelphia, but within two days the symptoms disappeared.

The second case was that of a patient who got a small piece of peanut in the lung. I did a bronchoscopy and did not obtain the peanut, but the next day the patient coughed it up.

In the third case, a long, thin nail which a carpenter was holding in his mouth was swallowed and went far down into a bronchiole. The longest tube that I had did not reach the nail. I put the patient to bed and thought that I would remove it in about three days. During that period nature threw a wall of thick mucus around the nail, and when the patient coughed, it flew out across the room. The patient recovered.

THE WEST OPERATION FOR DACRYOCYSTITIS. DR. CARL F. BOOKWALTER.

During the past twenty years I have performed one hundred and fifteen operations for the relief of chronic dacryocystitis, using what is practically the West operation; that is, making a permanent opening from the nose into the lacrimal sac by the intranasal route, without external incision. This method of treatment is indicated in cases of obstruction in the nasal duct but not in cases of epiphora due to obstruction of the canaliculi without dacryocystitis.

In cases of dacryocystitis with complete or partial obstruction of the canaliculi, the infection can be cured by this method, but no definite promise can be made about the relief of tearing. If the canaliculi are in reasonably good condition, there should be no epiphora after the operation.

I shall not describe the operative technic because this has been done often enough. There is a description with drawings in my article published in the *Archives of Ophthalmology* (**49**:568 [Nov.] 1920). One point should be strongly emphasized, namely, that the opening must be made as large as possible and by positive removal of tissue.

I do not know of any other otolaryngologic operation that gives so high a percentage of satisfactory results. In carefully selected cases the results should be good in nearly all instances. I do not advise this procedure for a young child because of the difficulty of working in a small nose. My youngest patient was 16 years of age.

One should be cautious about undertaking the operation in cases of dacryocystitis secondary to injury, as some unexpected difficulty may be encountered.

I have not done the Mosher operation. It may be easier to do than the West. The fact that no external incision is used is a point in favor of the West operation.

DISCUSSION

DR. G. HENRY MUNDT: When one considers the discussion of dacryocystitis given tonight and that given at the meeting a month ago one would think that the management of the condition was simple. It may be in the hands of some workers but not for all. As Dr. Bookwalter said, the person who has a canaliculus slit and not a round puncta will always have epiphora. I had some experience with the West operation several years ago, and I think that I had a fair proportion of good results, but the final word on surgical procedures for dacryocystitis has not been said.

DR. ROBERT BLUE: I wish to emphasize that dacryocystitis, whether acute or chronic, is most distressing to the patient. The chronic cases extend over many years, from one or two to twenty or thirty. In the cases in which the

West operation is indicated—and it is only indicated in certain cases, as Dr. Bookwalter mentioned—it gives satisfactory results. The West operation certainly is not indicated in the chronic cases, in those in the old persons or in the acute cases until they become subacute. I have seen the results of some of Dr. Bookwalter's operations, and from the patient's standpoint the results are most gratifying.

DR. ROBERT GOOD: I feel that I should emphasize one thing that Dr. Bookwalter brought out and that Dr. Mundt emphasized, namely, slitting of the canaliculus. I think that that should never be done, and I never do it. The trouble is below, not above. The West operation is not hard to do, and Dr. Bookwalter made an important point in regard to making the opening large. That is the only safe thing to do. I have performed a new operation twenty-seven times and have not had a failure. Epiphora has resulted three times, which could easily be removed. I do not wish to take time to describe this procedure tonight.

DR. WALTER H. THEOBALD: Will Dr. Bookwalter give briefly the technic of the West operation?

DR. CARL F. BOOKWALTER: Even in the cases in which there is a slit or obstruction in the canaliculus there is great improvement in respect to tearing and the patient is well satisfied. One of the important things about the operation is that the patient is satisfied. One of the chief difficulties is in securing patients, for nearly all of them go to ophthalmologists. The operation is not difficult. Dr. Blue pointed out that it should not be done in acute cases, and I brought that out in my paper.

It is rather difficult to describe the operation without the use of lantern slides. I could not do it very successfully just by talking about it.

ADJUSTMENT AFTER LABYRINTHECTOMY. DR. ELLISON L. ROSS.

One inner ear was destroyed in each dog of a series. Then each animal was put daily into a standardized whirling machine. Measurements of the duration of nystagmus following increases and decreases of pressure on the ampullae of the posterior, anterior and horizontal canals were made. These observations were made daily for a period of fifteen to eighteen days.

Spontaneous nystagmus was present for three or four days after the operation. Because the spontaneous nystagmus complicated the interpretation of the results obtained during the first four days, the averages for the fifth, sixth and seventh days were calculated. These averages give relative values for each canal. The standard increase of pressure on the ampulla of the horizontal canal produced nystagmus for 19.6 seconds. On the posterior canal it produced nystagmus for 10.8 seconds, and on the anterior canal, for 9.5 seconds. An equivalent decrease in pressure on the ampullae of the horizontal, posterior and anterior canals produced nystagmus lasting 10.0, 17.5 and 20.2 seconds, respectively.

These results indicate that an increase in pressure on the ampulla of the horizontal canal produces nystagmus of approximately twice the duration of that caused by an equivalent decrease. The opposite is true for the other canals; the decreased pressure produced the greater effects.

The direction of the nystagmus was the same for all the canals. An increase in pressure brought on nystagmus toward the same side, while a decrease induced nystagmus toward the opposite side. This led to a conclusion which differs from that of Ewald. It is commonly stated in textbooks that negative pressure in the horizontal canal on one side is inhibitory to the influence of increased pressure on the canal of the opposite side. One also reads that increased pressure on the ampullae of the vertical canals inhibits the reaction to the decreased pressure in the canals of the opposite side. My results indicate that a movement in any plane causes a reaction which is the sum of the effects of decreased pressure on one side and increased pressure on the other side.

Why does an animal regain its equilibrium after the destruction of the labyrinth on one side? Does the difference between the influence of the increased and that of the decreased pressure on the ampullae become less marked? Do the vertical canals come to react more like the horizontal canal?

To determine this point, my associates and I removed one labyrinth from each of ten dogs. Daily for fifteen days three tests were made on each dog for the response to negative and positive pressure changes on the ampullae of the horizontal, anterior and posterior canals. Toward the latter part of the period two or three dogs died, so that at the end there were not observations on the whole number of animals. Therefore, our average daily value for each canal is the result of thirty measurements.

The duration of nystagmus produced by a standard stimulation by increased pressure on the ampullae of the horizontal, anterior and posterior canals was charted.

Beginning with the fourth day after operation, stimulation of the horizontal canal produced nystagmus lasting 22 seconds. By an irregular decline, the nystagmus reached a duration of about 17 seconds on the fifteenth day.

Pressure on the anterior canal on the fourth day produced nystagmus for a little less than 11 seconds, and on the fifteenth day the duration was a little over 10 seconds. The irregular sagging of the curve probably has no significance.

Pressure on the posterior canal on the fourth day produced nystagmus for 11 seconds, and at the end of fifteen days the duration was a little less than 10 seconds. The change is considered insignificant.

Therefore, it must be concluded that there is no readjustment of the vertical canals in their response to increase of pressure on the ampullae. It is also concluded that there is an adjustment of the horizontal canal; the sensitiveness to increased pressure on the ampullae decreased.

A chart indicates the duration of nystagmus after a standard decrease in pressure on the ampulla of each canal and the variation over a period of fifteen days.

Decreased pressure on the horizontal canal on the fourth day produced nystagmus lasting a little over 10 seconds. On the fifteenth day the duration was nearly 10 seconds. The change was insignificant.

Decreased pressure on the anterior canal on the fourth day produced nystagmus lasting over 19 seconds, and on the fifteenth day the duration was a little over 17.5 seconds. The difference between the values for the fourth and fifteenth days was not as great as the general fall of the curve indicated. The average duration for the fourth, fifth and sixth days was approximately 20 seconds, and the average for the thirteenth, fourteenth, and fifteenth days was 17 seconds. This indicates more truly the decline in the sensitiveness of the anterior canal.

Decreased pressure on the posterior canal on the fourth day produced nystagmus for nearly 18 seconds, and on the fifteenth day the duration was 13 seconds. This difference, combined with the decided general fall of the curve, indicates that there was a decided decrease in the reaction caused by the stimulation of the posterior canal.

Therefore, our animals showed decided decreases in reaction to increased pressure on the ampullae of the horizontal canals and to decreased pressure on the vertical canals. The observations also show that there was no change in the reaction as a result of decreased pressure on the ampullae of the horizontal canals or of increased pressure on the vertical canals.

The charts also show that in fifteen days the reaction to increased pressure never equaled the reaction to decreased pressure in any one canal.

The problem of readjustment of equilibrium after complete or partial destruction of one labyrinth involves many factors. The changes in the irritability of the ampullae are but one. We have made observations on this adjustment as influenced by the posterior columns of the spinal cord. We are now attempting to evaluate the rôle of the cerebellum in the readjustment. There is much evidence to indicate that the cerebrum, more than any other influence, modifies the vestibular reaction.

DISCUSSION

DR. OTIS F. MACLAY: I congratulate Dr. Ross on his work. It has taken a long time and a considerable amount of hard work with a very careful checking up on the findings. What he has found has given definite information in regard to the canals.

DR. ALFRED LEWY: The findings reported by Dr. Ross, if I understand him correctly, seem to support the Mach-Breuer and Ewald hypothesis and have been checked by various experiments in which the result of the impact or acceleration of the endolymph has been observed. These experiments consisted of introducing a fine cannula into the semicircular canals and applying pressure. Dr. Ross' terminology is somewhat different from that generally used when he speaks of negative and positive stimulation. I have been accustomed to assume, on an experimental basis, that when the acceleration of the fluid in the horizontal canal is from the canal toward the utricle a greater stimulation is produced than by the reverse movement, and that in the superior canal (which Dr. Ross calls the anterior) the greater stimulation results when the movement is from the utricle toward the canal. Dr. Ross' experiments seem to support this.

DR. SHERMAN SHAPIRO: Dr. Ross' paper opens up a complicated subject. Dr. Lewy stated some of the things I intended to say and expressed my thought when he said that these experiments are valuable mainly in corroborating the findings of Ewald.

I was interested and somewhat surprised to hear Dr. Ross talk about stimulation and depression with respect to labyrinthine phenomena. This is an old theory of labyrinthine action which has been almost abandoned because of certain facts which cannot be explained except on the basis of a current in the canals. To cite a familiar example, if one irrigates an ear with the head 60 degrees back and after the proper nystagmus is obtained has the patient bend 120 degrees forward the direction of the nystagmus is reversed. Maier and Lion, and more recently Schmaltz, have also demonstrated the plausibility of a current or at least some type of endolymph movement on an experimental basis.

As to rapid approximation of the duration of nystagmus following rotation to each side in animals who have been subjected to unilateral labyrinthectomy, I believe that the results given by Dr. Ross correspond fairly well with the clinical observations on patients suffering from acute suppurative labyrinthitis. There is one aspect of this so-called "compensation" that I should like to mention. It is known that the permanent effects of destruction of the labyrinth vary with the species of animal in question. For example, the pigeon becomes unable to fly following such an operation on both sides, and even unilateral removal causes considerable difficulty in this respect, while in higher animals the effects of such procedures are increasingly less marked. In man, one has been accustomed to consider that exclusion of a labyrinth is followed by compensation within a week or ten days. I have been able to demonstrate by means of Frenzel's illuminated glasses spontaneous nystagmus several months after the effects of the labyrinthitis had apparently disappeared. This brings up the question whether such persons are justified in working under conditions which call for a perfect sense of equilibrium, as the "compensation" which has taken place is probably never complete.

DR. SAMUEL J. PEARLMAN: A number of years ago Griffith, at the University of Illinois, demonstrated that repeated rotations ultimately have an effect on nystagmus. I should like to know what Dr. Ross thinks of the factor of habituation following repeated rotation in connection with his experiments.

DR. ELLISON J. ROSS: In relation to the remarks of Dr. Shapiro, I do not believe that over a period of a month, or even two or three months, the reactions to positive and negative stimulations would become equal.

We tried daily rotations for a number of days on a number of animals that had not been operated on and a number that had undergone operations and did not find any considerable fall in the duration of nystagmus. There is a small fall for the first three or four days, but after that the response is just as before.

MOTION PICTURES OF EYES IN CASES OF LESIONS OF THE BRAIN. DR. J. GORDON WILSON.

Book Reviews

Chronic Nasal Sinusitis and Its Relation to General Medicine (Chronic Sinusitis and Systemic Sepsis). By Patrick Watson-Williams. With a foreword by Sir Humphry Davy Rolleston. Second edition. Price, $5. Pp. 262, with 123 illustrations. Baltimore: William Wood & Company, 1934.

The first edition of this book appeared in 1930. It is reviewed in the ARCHIVES OF OTOLARYNGOLOGY (**12**:553 [Oct.] 1930). The present edition has 262 pages —41 pages more than the first edition. The volume is well supplied with illustrations. It is primarily a plea in support of the thesis that foci of infection in the nasal sinuses are a frequent source of systemic disease. The diagnosis of sinus disorders is carefully gone over, and illustrative cases are interspersed to support the author's hypothesis.

On the whole, the author deserves credit for the careful manner in which he has carried on his work, but he appears to be on rather disputable grounds when it comes to the application of the hypothesis of focal infection to cases in which the recognition of sinus disease cannot be made from the usual local findings. In these cases the author relies on his method of withdrawing the contents from the suspected sinus by suction, resorting to the introduction of fluid and its withdrawal for examination. When palpable evidence of pathologic material is not revealed to the naked eye, he would resort to centrifugating the fluid withdrawn, in order to discover cellular elements on which to base the diagnosis.

Macmillan Medical Monographs. George R. Minot, Editorial Advisor. **The Medical and Orthopaedic Management of Chronic Arthritis.** By Ralph Pemberton and Robert B. Osgood. Price, $5. Pp. 403, with 102 illustrations. New York: The Macmillan Company, 1934.

"This book has been written because of the convictions of the authors that chronic arthritis is largely a preventable and curable disease; and that current knowledge of the nature of the disease and of the measures necessary to combat it is not as generally diffused throughout the medical profession as it should be. . . . The effort has been made to present a perspective of the syndrome of chronic arthritis in which etiology, pathology, physiological disturbances, and treatment of the disease are seen in their respective relations one to another as constituting a whole." The treatise impresses one as a thoroughly sane and logical presentation of the history, classification, pathology and physiologic disturbances of this clinical entity. The etiology and symptomatology are discussed, as well as the various phases of the treatment: medicoclinical treatment, treatment of focal infection, treatment by the gastro-intestinal route and treatment with vaccines. The authors have throughout preserved a judicious poise in the discussion of the several problems, a poise which is much needed in these days when the complexity of medical problems has led to an intensive development of fads.

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